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IRIDENCLEISIS IN BUPHTHALMOS

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Buphthalmos, or juvenile glaucoma, in the past presented an exceedingly gloomy prognosis. A questionnaire sent by Patton,[1] in 1925, to a number of active American ophthalmologists revealed that 45 per cent were advising no treatment for the condition, except enucleation when indicated for pain; 27 per cent employed nonsurgical treatment, but reported no favorable results.

The same impression was gathered by Blake[2] from a similar questionnaire. Notes on 472 cases were obtained from 178 ophthalmologists. Only 156 operations had been done, including 78 iridectomies, 50 corneoscleral trephinings, 15 anterior sclerotomies, 10 sclerectoiridectomies according to Lagrange and 3 iridotases. Blake reported no personal cases, but advised trephining during the first year of life. In answer to Patton's inquiry only 1 man, A. L. Beck, reported success with iridectomy. (Most operators who tried iridectomy were disappointed, and before the advent of the filtering operations this accounted for the stand taken by many ophthalmologists against operation in buphthalmos.) John Green, Sr., Lancaster, Peter, Robertson, Bedell, Ellett and Feingold were encouraged by their results with trephining. From the practice of H. Gifford, Patton and myself, Patton reported 13 cases; these showed 1 good result from sclerotomy and trephining, 1 fair result with anterior sclerotomy and subsequent puncture of a prolapsed iris, 1 failure from iridotasis and 2 successes following iridencleisis, 1 of which will be described in the present report. A number of cases were seen late when no vision was present, and in these no operation was advised. Patton made a plea for early surgical intervention in these cases, advising one of the filtering operations. Fleischer,[3] in 1918, listed the negative results obtained with trephining by Bjerke, Casey Wood and H. Gifford, and opposed to this the good results of Zeemann (in 6 of 11 eyes), Grunert (in 4), Dubois (in 3) and Clegg (in 1). The results in his own series from the Tübingen

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1. Patton: Tr. Am. Acad. Ophth. **30**:38, 1925.
2. Blake: Arch. Ophth. **54**:1, 1925.
3. Fleischer: Klin. Monatsbl. f. Augenh. **61**:152, 1918.

Clinic were very favorable for trephining. No bad results were obtained, and only once was vitreous lost. In 7 of 11 cases the tension was permanently reduced to normal. In the 7 eyes which were operated on during the first year of life damage to the eye seemed to have been avoided entirely, as the nerves were normal and vision was apparently excellent. He commented on the smooth healing of the trephine scars, with no apparent fistulization or bleb, and considered the danger of late infection negligible. He also observed several cases in which the tension returned slowly to normal, requiring treatment with pilocarpine for several months to reach that level. In only 2 cases was a second trephining performed; good results were obtained in all.

Poulard and Lavat [4] reported favorably on the effect of trephining and the Lagrange sclerecto-iridectomy. The former was performed 16 times, and the latter twice, on 18 eyes (11 patients). In two thirds of the patients the tension was permanently reduced to 28 mm. (Schiötz) or less. Six patients operated on within the first year of life had apparently excellent vision.

Dettmering [5] recently reported 55 cases from the Göttingen Clinic, 45 eyes being operated on. Of 23 eyes trephined at the primary or secondary operation 6 showed a good result (vision 1/10 or better) and 4 a partially good result; in 13 the operation was unsuccessful. Repeated sclerotomies were performed in most of the others, with 1 good and 3 partially good results and 3 failures.

Goerlitz [6] trephined 4 eyes with reduction of tension in all, and Wright [7] reported 2 successful trephinings in cases developing in late childhood. Spratt [8] reported vision in one eye amounting to ability to count fingers seventeen years after trephining although the tension was 36 mm. (Schiötz). The other eye became phthisical following operation at the same time.

Against these encouraging results with trephining are to be placed the poor results previously referred to. H. Gifford [9] reported 2 failures to reduce tension, the bleb healing over so firmly that no filtration occurred. He believed this was due to the large size of the eye, which held the conjunctiva firmly in contact with it and so prevented the formation of a bleb. He suggested placing the trephine opening as far back as possible while entering the anterior chamber, as he believed

4. Poulard and Lavat: Ann. d'ocul. **162**:496, 1925.

5. Dettmering: Inaug. Dissert., Göttingen, 1932; abstr., Ztschr. f. Augenh. **81**:271, 1933.

6. Goerlitz: Klin. Monatsbl. f. Augenh. **74**:778, 1923.

7. Wright: Brit. J. Ophth. **15**:166, 1931.

8. Spratt, C. N.: Trephining for Buphthalmos, Arch. Ophth. **4**:338 (Sept.) 1930.

9. Gifford, H.: Ophth. Rec. **25**:462, 1916.

the conjunctiva to be looser in this area. Others have noted loss of vitreous and other complications resulting in loss of the eye. Zeemann, in spite of the good results referred to, lost 3 eyes, by excessive loss of vitreous, late detachment and late infection, respectively, and others have reported similar experiences due to sudden reduction of tension in eyes with an atrophic zonula and in some cases as a result of fluid vitreous.

Thus, while trephining has undoubtedly resulted in saving many eyes, it is more liable to certain serious complications in buphthalmos than in chronic simple glaucoma.

For this reason I tried iridencleisis in a case of the juvenile form of buphthalmos, and my success led me to employ it in the only cases I have seen since that time which were suitable for operation. Holth mentioned its advantages in this condition, though he gave no reports of cases, and Verhoeff,[10] in describing his modified iridotasis, stated that it had been successful in buphthalmos and was particularly indicated in that condition. Blaickner,[11] however, an advocate of iridencleisis in chronic glaucoma, believed it contraindicated in buphthalmos. The success of iridencleisis in my small series of cases leads me to report them in the hope that others may be led to try the procedure.

REPORT OF CASES

Case 1.—Everett O. was seen in 1925 when 14 years old. Both corneas were 12 mm. in diameter, and the tension varied between 45 and 50 mm. (Schiötz). Vision was 20/200 in the right eye and 20/30 in the left, and the fields were reduced to within 15 degrees in both. Miotics reduced the tension for only a short period, and it rose to 45 in each eye. Iridencleisis was performed on both eyes. The tension was 27 in the right eye and 23 in the left soon after the operation; vision was 20/70—1 in the right eye and 20/30 in the left, and the visual fields showed no change. Tension remained normal in the left eye. At the last examination, in 1930, the tension was 18, vision 20/15 with correction and the field the same as before. The right eye showed a return of tension to 32 after three months, and though it was normal at most subsequent examinations, the field grew smaller; after six years vision was reduced to perception of hand movements, although tension varied between 25 and 21.5. At one examination in 1930 a tension of 48 was found, and there were presumably periods of increased tension which accounted for the loss of vision. In this eye a large ectatic scar, containing atrophic iris tissue, developed well back from the limbus, while in the left eye—in which the condition remained under control—the scar appeared only moderately elevated, was covered by thick conjunctiva and showed very slight evidence of filtration (fig. 1).

Case 2.—Robert M., aged 3 months, was seen in April, 1931. The child of a maternal uncle was known to have had one eye which grew large and ruptured, while the other eye remained normal. Except for the ocular condition the patient was healthy. Both corneas measured 13.5 mm. and were diffusely hazy with more

10. Verhoeff: Arch. Ophth. **56**:386, 1926.
11. Blaickner: Ztschr. f. Augenh. **72**:265, 1930.

opaque striae. Tactile tension was very high, and with lid retractors and fixation a reading of from 45 to 50 was obtained in each eye. Six days later an operation was performed on both eyes. The child had been seen in consultation with a very experienced colleague, and at the parent's request this surgeon operated on the left eye, doing a deep iridectomy, while I performed iridencleisis on the right eye at the

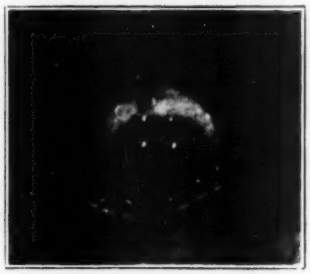

Fig. 1 (case 1).—Left eye six years after operation.

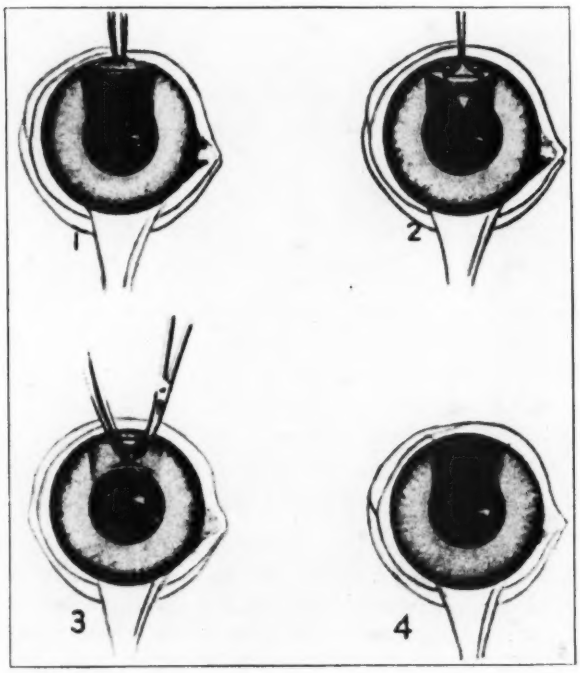

Fig. 2.—Technic of iridencleisis with running conjunctival suture.

same time. Both corneas cleared markedly after operation, and the right remained clear. The left cornea, however, became cloudy after several weeks, and after two months the iridectomy was repeated on that eye by the same colleague. Tension under anesthesia at this time was 25 in the right eye and 40 in the left. The pupil was pulled up and out in the left eye, and after four months the cornea was again slightly cloudy. An intermittent convergence of this eye was noted. Three months later tension under anesthesia was 28 in the right eye and 40 in the left.

but four months later both corneas were clear and tension under anesthesia was 25 in the right eye and 25 in the left. The right nerve was somewhat pale but not excavated, and the left fundus was difficult to see on account of a high degree of astigmatism. At the last examination, two years after operation, both corneas were quite clear, tactile tension was normal, and the child appeared to have excellent vision, picking up very small objects, walking well and playing like a normal child.

Case 3.—John M., when seen in April, 1933, at 10 months of age, had well developed buphthalmos. The corneas were large and extremely cloudy. Iridencleisis was performed at once, and at operation tension was found to be 72 in both eyes. The eyes cleared rapidly after operation, and a month later tension under anesthesia was 10.5 in the right eye and 22 in the left. Two months later the left cornea was clear but the right was cloudy; tension was 55 in the right eye and 19 in the left. Iridencleisis was repeated on the right eye, slightly above the horizontal meridian on the temporal side. At the last examination, on November 24, tension was 25 in the right eye and 14 in the left, and the corneas were clear except for one diagonal stripe in the right cornea. Vision seemed good, and the eyes were kept open normally. In both eyes of this child and in the right eye of the patient in case 2, small ectatic scars formed, covered by thick conjunctiva but with no evidence of filtration.

TECHNIC OF OPERATION

The operation is performed exactly as described in a previous paper,[12] Holth's technic being followed with the exception of the conjunctival flap. This is made through a horizontal incision just in front of the insertion of the superior rectus and the untied running conjunctival suture, the so-called "water-tight" suture of Foster Moore. Especially useful in this operation, which must be performed under general anesthesia, is fixation on the superior rectus through the intact conjunctiva with H. Gifford's fixation forceps, which have long teeth and a clamp. This is applied as the first step in the operation. The forceps is allowed to rest on the upper lid, in which position it exerts no pressure, as the lid acts as a fulcrum to elevate its attachment to the globe. The eye thus is held down during the subsequent procedures, and the forceps are removed only when the final suture is placed (fig. 2).

RESULTS

In this series of operations on 5 eyes (3 cases), 3 may be considered as completely successful. One operation, that on the right eye in case 3, may probably also be considered a success, as tension was 25 after a second iridencleisis, though the time is still too short for one to be certain. Another operation, that on the left eye in case 1, may be called a partial success, as vision was maintained for two years but subsequently failed; tension was normal.

No complications were encountered in the operations. Vitreous was not lost, nor was the lens injured. While one might not be so fortunate in a larger series of cases, it would seem that this freedom from complications sometimes resulting in loss of the eye would serve to recommend iridencleisis.

12. Gifford, S. R.: Tr. Am. Acad. Ophth. **32**:117, 1927.

It is difficult to explain the effect of this operation or trephining in buphthalmos, as usually almost no blebs are formed. It would seem that new avenues for the drainage of fluid under the conjunctiva remain open for a certain time and that perhaps the reduction of tension for this period breaks up a vicious circle resembling that of acute glaucoma, allowing other normal though previously insufficient avenues of drainage to be resumed gradually.

Certainly, with this procedure at his disposal and trephining as an alternative, the modern ophthalmologist is not justified in a policy of inaction when confronted with a case of buphthalmos, if vision is still present. Nor should he give a prognosis so gloomy that parents will be discouraged from submitting their child to a surgical procedure which offers the only prospect of preserving useful vision.

DIABETIC AND TOBACCO AMBLYOPIA

REPORT OF A CASE

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A person may be a heavy drinker and smoker for years and never notice any depreciation of vision. A diabetic person may go through life and suffer no visual disturbance. However, if the heavy smoker and drinker acquires diabetes, this relative immunity is apt to change. The report of the following case exemplifies this apparent relationship between diabetes and toxic amblyopia.

REPORT OF CASE

Mr. H. F., aged 42, was first seen on May 3, 1933, with the complaint of poor vision in both eyes. The onset was sudden, beginning three months before consultation. At this same time, the patient also learned that he had diabetes. He had been a heavy drinker and smoker for over fifteen years, consuming about a pint of whisky and smoking from fifteen to twenty cigars a day. The vision had apparently been normal all this time until about three months before consultation, when the diabetes appeared.

Ocular examination revealed vision of 6/60 in the right eye and 6/22 in the left eye. External ocular examination gave normal results. The temporal half of the right disk was pale; the left fundus seemed normal. Studies of the peripheral fields showed no contraction for form or color. In each eye, there was a cecocentral scotoma for blue, green and red. The scotoma was more dense in the right eye (chart 1). A diagnosis of toxic amblyopia complicated by diabetes was made, and the patient was admitted to the Jefferson Hospital.

General physical examination gave negative results except for infected tonsils. The blood studies were normal. Urinalysis revealed a large amount of acetone.

The patient was placed on a diabetic regimen, supplemented by 10 units of insulin daily. Tonsillectomy was performed, and general eliminative treatment was carried out. The patient was advised to discontinue the use of alcohol and tobacco. In addition, he was also given weekly intravenous injections of sodium nitrite to which had been added other inorganic salts. The improvement was slow but progressive. The diabetes was controlled promptly, and the acetone disappeared rapidly. The scotomas began to grow less dense and retract from fixation, receding gradually toward the blind spot. The patient was discharged from the hospital three months after admission with a vision of 6/9 in each eye. In the left eye, the scotoma had completely disappeared; in the right eye, there was a small finger-like projection of the blind spot extending toward fixation. The final studies of the fields, made on subsequent observation, are shown in chart 2.

REVIEW OF LITERATURE

According to some authors, the presence of amblyopia in diabetes is common; in the opinion of others, it is unusual. Schmidt-Rimpler [1] found amblyopia in 34 of 140 cases of diabetes, a rather high percentage. Kako,[2] on the other hand, saw it in only 10 of 240 cases, and Foster Moore [3] observed it in 6 of 60 cases. Von Noorden [4] repeated Uhthoff's

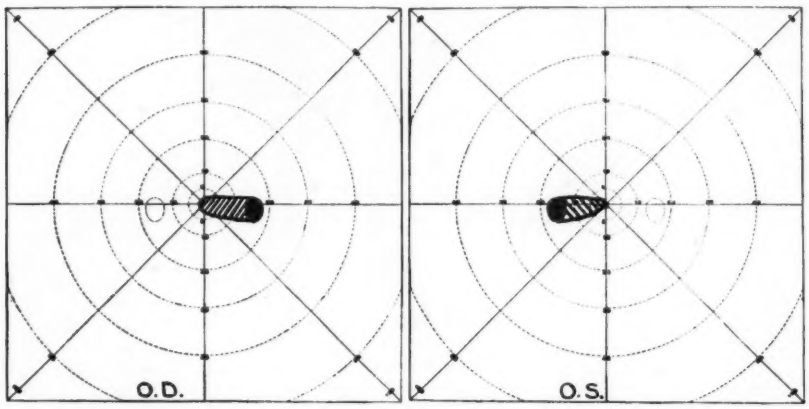

Chart 1.—Visual fields taken on May 1, 1933. In the right eye there was a cecocentral scotoma for red, green and blue with a 2 degree test object.

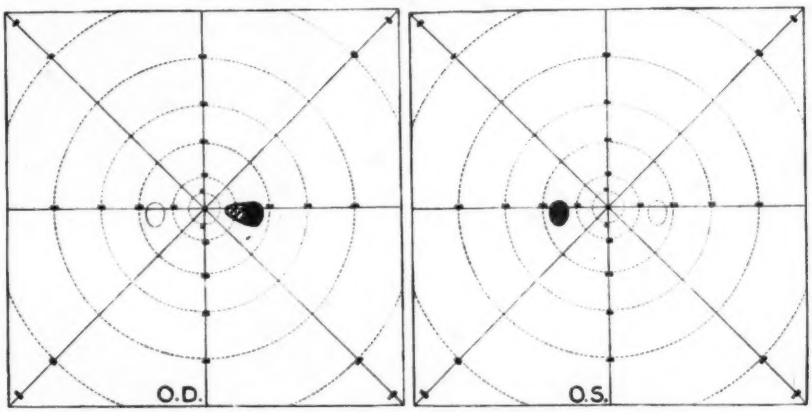

Chart 2.—Visual fields taken on Sept. 11, 1933.

1. Schmidt-Rimpler: Ber. d. ophth. Gesellsch., Heidelberg **25**:99, 1896.

2. Kako, M.: Beiträge zur Kenntnis der Augenaffektionen bei Diabetes Mellitus, Klin. Monatsbl. f. Augenh. **41**:269, 1903.

3. Moore, R. F.: Medical Ophthalmology, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1924, p. 170.

4. von Noorden, C. H.: Die Zuckerkrankheit und ihre Behandlung, Berlin, Julius Springer, 1927, p. 327.

figures of 16 cases of amblyopia in 280 persons with diabetes. Leber's [5] 14 cases in 50 diabetic persons are also frequently quoted. In the literature both in America and on the Continent, few cases have been reported since 1900.

The disease predominates in the male sex. In the combined cases reported by Leber,[5] Jensen, Kako,[2] Blegvad and Rönne,[6] a total of 48, only 1 woman was affected. For this reason, Rönne believes that the condition resembles Leber's disease.

The complication of tobacco and alcohol plays an important rôle in this disease. All the writers on this subject have mentioned this point. Galezowski [7] appears to have been the first to draw attention to the relationship between diabetic and tobacco amblyopia. He believed that diabetic amblyopia was inclined to be unilateral, whereas tobacco amblyopia was usually bilateral. This is not borne out by the literature. The majority of cases of diabetic amblyopia have been bilateral.

The presence of the scotomas and their shape, position and character have been excellently described by de Schweinitz and Fewell.[8] The literature on this subject up to 1913 was reviewed by Wilbrand and Sänger.[9] All admit that the scotoma in diabetes and in tobacco amblyopia is peculiar to each disease. The scotoma may be unilateral in diabetes. Wilbrand and Sänger reported 2 such cases. The peripheral fields are usually normal, although in the cases of Fieuzal and Samuel, there was a contraction. The scotomas are usually relative, although Kako saw 2 cases of absolute scotoma in 7 persons with diabetes. Foster Moore reported 1 case.

The fundi are usually normal. In the cases of Wilbrand and Sänger, Kako, Laquer, Nettleship and Edmunds,[10] Zirm and Dimmer,[11] there was pallor of the temporal half of the disk.

The relationship of tobacco to the production of a scotoma in a diabetic person is not an academic one. Moore [12] had already stated

5. Leber: Ber. d. ophth. Gesellsch., Heidelberg **25**:99, 1896.

6. Rönne, H.: Zur pathologischen Anatomie der diabetischen Intoxikationsamblyopie, Arch. f. Ophth. **85**:489, 1913.

7. Galezowski: Des amblyopies et des amauroses toxiques, Rec. d'opht. **5**:104, 1878.

8. de Schweinitz, G. E., and Fewell, A. G.: Therap. Gaz. **50**:623, 1926.

9. Wilbrand, H., and Sänger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1913, vol. 5, p. 130.

10. Nettleship, E., and Edmunds, W.: Two Cases of Symmetrical Amblyopia with Slow Progress with Central Scotoma in Patients Suffering from Diabetes; Microscopic Examination of a Nerve in One Case, Tr. Ophth. Soc. U. Kingdom **1**:124, 1881; On Central Amblyopia in Diabetes, Especially as to Its Dependence on, or Independence of, Tobacco Smoking, ibid. **3**:165, 1883.

11. Dimmer: Klin. Monatsbl. f. Augenh. **39**:570, 1901.

12. Moore, W. D.: Diabetic Affections of the Eye, New York M. J. **47**:339, 1888.

that amblyopia occurring in diabetic persons who were heavy smokers is due to the loss of nutrition produced by the diabetes, and not directly to the action of the toxin from the tobacco. Many years later, Holth [13] expressed a similar opinion. He believed that the diabetes lowered the general tolerance, causing the development of the scotoma. As soon as the diabetes is controlled, the tolerance to nicotine is raised and the scotoma disappears.

Nettleship and Edmunds [10] believed that diabetes acted as an influence in predisposing those who smoke to disease of the optic nerve. Leber, in discussing Schmidt-Rimpler's paper, remarked that it was not easy to distinguish diabetic amblyopia from tobacco-alcohol amblyopia. He also stated that a person with diabetes is more susceptible to nicotine poison than a normal person. He added that the improvement seen in his patient came from the diabetic treatment. Uhthoff,[14] at this same meeting, held that diabetic and tobacco amblyopia could not be differentiated. This statement is not accepted by any later writers on this subject. Traquair,[15] quoting Rönne, held that tobacco amblyopia produces a definite scotoma, cecocentral in type, which is not found in any other disease.

According to de Schweinitz and Fewell, the scotomas of tobacco amblyopia have distinctive features which serve in most instances to distinguish them from other toxic scotomas in the central area of the field. The scotoma is cecocentral with sloping margins, never truly central, and often contains absolute areas within the relative area. The authors reported 3 cases showing these features. They agree with Traquair, stating that in cases of general disease, such as diabetes, in smokers the amblyopia should be attributed to the tobacco if the scotoma is typical.

Rönne believed that the diabetic amblyopia develops more suddenly and is more intense than tobacco amblyopia.

The actual cause of the amblyopia in persons with diabetes who smoke is not known. Gallus believed that the acidosis is responsible for its production. Rönne [16] recently expressed a similar idea, stating that the acetonemia may be the cause of the lowered vision. Francis and Koenig [17] previously suggested this same explanation. In most of

13. Holth, S.: Amblopie centralis eines diabetischen Kattenrauchers, geheilt unter Insulinbehandlung trotz Wiederaufgenommenen und fortgesetzten Nikotinismus, Acta ophth. **5**:195, 1927.

14. Uhthoff: Tr. Internat. Cong. Med., 1900, p. 126

15. Traquair: Tr. Ophth. Soc. U. Kingdom **50**:351, 1930.

16. Rönne, H., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 714.

17. Francis, L. M., and Koenig, I.: Retrobulbar Neuritis in Diabetes, J. A. M. A. **87**:1373 (Oct. 23) 1926.

the cases of this disease reported in the literature there was acidosis. In the case here reported, the patient showed a large amount of acetone in the urine. One of Kako's cases resembled ours. The patient had been a heavy drinker and smoker for years. Traumatic diabetes developed, followed by amblyopia. Under diabetic treatment, the scotomas disappeared. On the other hand, Mauthner[18] reported 2 cases of diabetic amblyopia in smokers which did not improve until smoking was stopped. Holth recently treated a smoker with diabetic amblyopia with excellent results, even though the patient continued to smoke.

The pathologic reports of diabetic and tobacco amblyopia are few. About eight papers have been written on this subject, and, with the exception of Rönne's paper, most of them are short and incomplete. Rönne, using a Marchi stain, found no evidence of inflammation in the optic nerve. There was, on the contrary, a degeneration of the nerve fibers of the papillomacular bundle. He believed with Dalen that the old conception of Samelsohn, that of a primary interstitial neuritis, can no longer be held. The picture is one of degeneration rather than inflammation. Another important finding was the degeneration of the ganglion cells of the retina. Rönne stated that there are two types of ganglion cells in the retina, small ones in the center and larger ones in the periphery of the retina. It is the degeneration of these small cells in the central area which gives rise to the development of the relative scotoma. However, this idea was not new with Rönne. In 1900, Nuel,[19] at the International Congress of Medicine in Paris, expressed a similar idea. He maintained that the origin of the retrobulbar neuritis was twofold, in the nerve and in the retina. In his attempt to explain the peculiar susceptibility of the papillomacular bundle to this toxin, he quoted the explanation of Edinger, namely, that the fiber tract which is most used is the most sensitive to the action of the toxin.

Uhthoff, in discussing it, rejected this theory of retinal degeneration with ascending atrophy on the grounds that the peculiar shape of the scotoma, its negative character, the late appearance of the temporal pallor and the absence of any disturbance of the light sense speak against a retinal origin in this disease. Regarding the fine pigment changes seen between the disk and the macula, Uhthoff held them to be of no value, since they are often found in normal fundi. He believed that the cause of amblyopia was inflammatory, beginning as an interstitial optic neuritis and resulting in a descending optic atrophy. This was held by Schmidt-Rimpler, Nettleship and Edmunds and Wilbrand and Sänger.

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18. Mauthner, L.: Amblyopia diabetica, Internat. klin. Rundschau **7**:209, 1893.
19. Nuel: Tr. Internat. Cong. Med., 1900, p. 126.

Parinaud,[20] at this same meeting, refused to take sides. He suggested that there may be a central lesion in the brain to explain the sudden onset, the symmetrical character of the scotomas and the disturbance of the color fields. All these, in his opinion, could not be explained by a lesion of the papillomacular bundles alone.

Abelsdorf [21] recently reported his findings in a case of optic atrophy in a person with diabetes of long duration, complicated by arteriosclerosis. He did not believe that the changes in the vessels had anything to do with the optic atrophy. In his opinion, the diabetes caused both the optic atrophy and the arteriosclerosis. His sections showed marked atrophy of the papillomacular bundle, extending beyond and including over half of the nerve. The medullary sheaths were almost completely absent. A few bundles of glial tissue and small round cells were scattered throughout the specimens. There was no definite sign of inflammation present.

CONCLUSION

As to the cause of diabetes in this patient, we can only hazard a guess. Lichwitz [22] and von Noorden believe that persons suffering from alcoholism are very susceptible to diabetes. We believe that a case reported here is one of toxic amblyopia, precipitated in some way by the onset of diabetes. Remembering the words of Foster Moore, that "there can be no doubt that diabetics are more vulnerable to the toxic effects of smoking than are healthy patients, and it is difficult at times to determine which of the two, the diabetes or the tobacco is the real factor in the production of the neuritis," it becomes our duty to warn persons with diabetes of the dangers of drinking and smoking.

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20. Parinaud: Tr. Internat. Cong. Med., 1900, p. 126.

21. Abelsdorf: Arch. f. Augenh. **95**:143, 1925.

22. Lichwitz, in von Bergmann, G., and Staehelin, R.: Handbuch der inneren Medizin, ed. 2, Berlin, Julius Springer, 1926, vol. 4, p. 337.

RETINITIS PUNCTATA ALBESCENS AND RETINITIS PIGMENTOSA AS AFFECTED BY PREGNANCY

REPORT OF A CASE

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The rare occurrence of retinitis punctata albescens, either alone or associated with retinitis pigmentosa, seems in itself sufficient excuse for reporting an additional case. There is also the further interest occasioned by the complication of pregnancy and the marked effect which the pregnancy apparently exerted on the course of the disease.

A thorough search of the literature fails to reveal any report of the effect of pregnancy on the course of this disease.

Vandergrift [1] stated that "undoubtedly there are lesions of the fundus to which the gravid uterus adds no further interest, such as retintitis pigmentosa, though even here arises the problem of the propagation of an hereditary maladjustment." Gifford [2] observed a case of retinitis pigmentosa in which pregnancy developed but in which no untoward effects on the retinal condition were observed.

However, there seems to be no reason why this ocular lesion should not be just as sensitive to the effects of pregnancy as are other diseases of the eye; as Black [3] has said, "It must be borne in mind that any serious eye lesions may be made worse by subsequent pregnancy."

It may be well to review briefly the etiologic factors as cited in cases already reported.

All authorities agree on the prominent part which heredity plays in the development of both retintitis punctata albescens and retinitis pigmentosa. Consanguinity also is frequently found. Thus, Nettleship,[4] in his remarkable observations on these conditions, found that of nine hundred and seventy-six families in which retinitis pigmentosa occurred, 23.5 per cent gave evidence of heredity without consanguinity, 23 per

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1. Vandergrift, G. W.: The Fundus Oculi in Pregnancy, M. Rev. of Rev. **35**:171, 1929.

2. Gifford, Sanford: Personal communication to the author.

3. Black, N. M.: A Résumé of Ocular Manifestations in Pregnancy, Wisconsin M. J. **20**:181, 1921.

4. Nettleship, E.: On Retinitis Pigmentosa and Allied Diseases, Roy. Lond. Ophth. Hosp. Rep. **17**:151, 1907-1908.

cent gave a history of consanguinity without a hereditary history and from 3 to 4 per cent gave a history of both heredity and consanguinity. Hansell,[5] in an article on atypical retinitis pigmentosa, noted a report by Herrlingers of ninety-two cases, in forty-five of which a hereditary influence was shown.

Some authorities, however, believe that consanguinity has been given too prominent a place in the etiology. They cite[6] the frequency with which it is found among the Hindus in whom consanguineous marriage is prohibited by religion.

When heredity is a factor, the disease appears early in life; when it is acquired, it appears later. Levine,[7] in a recent article, again advanced the idea that deficiency in vitamin A may be responsible for this retinal manifestation. This is in line with certain forms of treatment which have been recommended for many years. Many causes are listed for those cases which are considered to be of the acquired type. Of these, syphilis, typhus fever, pellagra, rickets and cirrhosis of the liver may be mentioned. In 50 per cent, no exciting cause could be determined.[5]

Nettleship[4] thus summarized his observations on the exciting causes of retinitis pigmentosa: "Such general causes as toxic illness and loss of blood may be supposed capable of operating either by lowering the power of resistance to an aready existing dormant tendency, or by inducing independent vascular disease, and probably both processes may sometimes be at work together." He cited a case in which severe uterine hemorrhage was followed by the development of retinitis pigmentosa.

Acute exanthems and other illnesses have also been cited either as the cause of the retinal disease or as accelerating the progress of one already in existence. Thus, Nettleship referred to the case of a family of ten children, five of whom had scarlet fever in early life followed by the development of retinitis pigmentosa; two others of this family died of the fever; three escaped the fever and did not have retinitis pigmentosa. This author then gave references from the literature to a long list of diseases following which retinitis pigmentosa developed: typhus fever, typhoid fever, scarlet fever, influenza, measles, etc.

The apparent relationship between these infections and the subsequent development of retinitis punctata albescens or retinitis pigmentosa

5. Hansell, H. F.: Atypical Retinitis Pigmentosa, Am. J. Ophth. **8**:374 (May) 1925.

6. Wood, C. A.: Retinitis Pigmentosa and Retinitis Punctata Albescens, American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1919, vol. 15.

7. Levine, Joseph: Retinitis Pigmentosa Due to Vitamin Deficiency, Arch. Ophth. **9**:453 (March) 1933.

is stressed in this report principally for the reason that, in the case to be reported, an acute infection is the only clue to an etiologic factor.

The following description of the fundus as found in my patient is similar, in general, to other descriptions of these conditions. The posterior portion of the fundus of both eyes is covered with small white dots. These are found in both the temporal and the nasal sides of the fundi, but do not involve the macular areas. Throughout the area in which the white dots predominate are found several pigmented areas. These consist of round spots of pigment approximately four or five times the size of the white dots.

In the periphery of the fundi are found still more numerous, round, pigmented areas. Here also are found branching deposits of pigment which have been described as resembling bone corpuscles. All of the blood vessels and more especially the arteries appear diminished in caliber, and toward the periphery they become narrow and threadlike. The optic nerve does not show any signs of atrophy.

These observations fit the picture of the fundi as seen in my patient over a period of approximately three years. Recently, however, further study of this patient shows a decided change in the appearance of the fundi. There is a marked increase of pigment both in the periphery and in the posterior areas of the fundus. In some of the areas which were covered with the white dots, the latter are now almost entirely replaced by the pigmented dots of approximately the same size, except that in numerous areas there appears to be a coalescence of the small dots into the larger, irregularly rounded spots of pigment.

Branching deposits of pigment are now much more numerous in the peripheral portions of the fundi and are found extending past the equator into the posterior fundi. In many places toward the periphery, the arteries appear to have been entirely covered by pigment.

REPORT OF CASE

Miss F., aged 17, came to my office on Aug. 5, 1929, complaining of not feeling well for the past two weeks. She stated that her throat had been sore during this time but had become much worse during the past few days.

Examination at this time revealed a patient acutely ill; her temperature was 102 F., and her pulse rate, 110. The tonsils were swollen and covered with grayish-white spots, presenting the typical appearance of Vincent's angina. Laboratory examination of the exudate showed large numbers of the fusiform bacilli and spirilla. The patient was treated at her home, and the infection in the throat soon cleared.

About two weeks later she again came to the office complaining of some difficulty in seeing well at night. Careful inquiry into her history failed to elicit any previous complaint of this nature. She had never had any previous trouble with reference to the eyes. The family history was also entirely irrelevant. There is

no consanguinity of the parents and no serious ocular complaint on either side of the family as far back as could be determined.

Central vision at this time was normal, and examination under mydriasis showed only a moderate degree of hyperopic astigmatism. Examination of the fundi revealed no definite changes, but the field of vision showed a slight contraction of the temporal field.

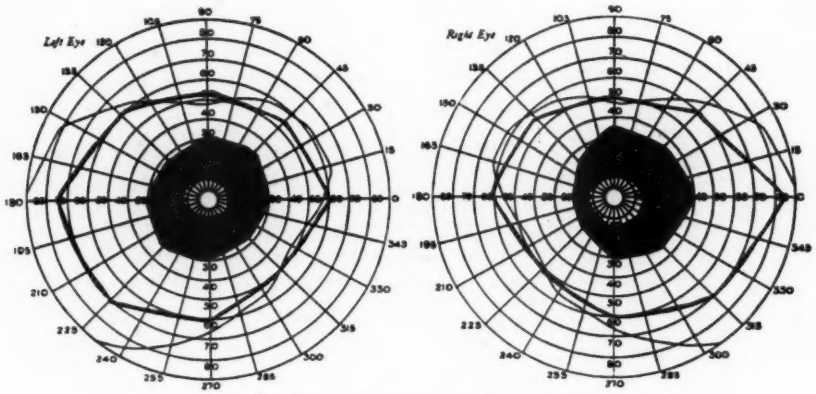

Chart 1.—Visual fields on Oct. 18, 1929.

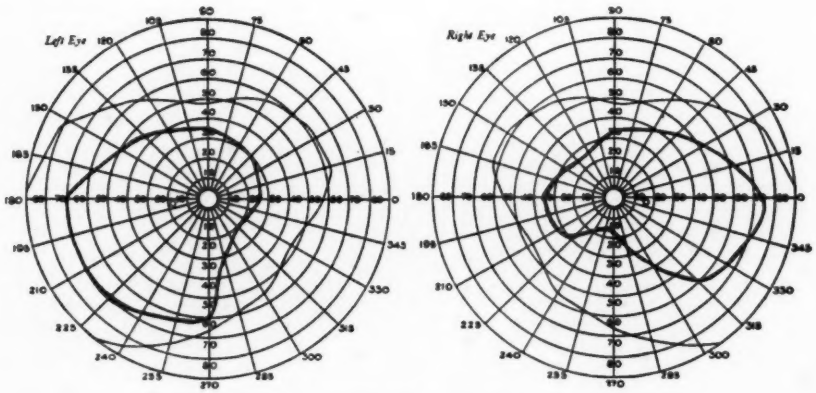

Chart 2.—Visual fields on Nov. 3, 1931.

The patient returned in about a month complaining of increased difficulty in seeing well at night, so that she was practically unable to go about alone. This was the middle of October, 1929, and an examination of the fields of vision showed a well defined scotoma (chart 1) with little change in the peripheral extent of the fields.

The Wassermann reaction of the blood was negative.

Central vision continued to be normal. One month later, that is, in the middle of November, an examination of the fields showed the ring scotoma still present in all but the nasal portions of the field. In the nasal sectors, there was a gradual clearing of the scotoma. By January, 1930, the scotoma had almost entirely disappeared, there being left only some blurring of the test target in certain portions

of the field. The patient was also quite happy in the fact that she was able to go about much better at night.

She was not seen from January, 1930, until December, 1930, a period of eleven months. The fields of vision taken on the later date showed only a slightly contracted form field with slight blurring of the target in certain portions of the field.

Again, from December, 1930, to November, 1931, the patient was not seen. During this time she had been married. On Nov. 3, 1931, she came to the office stating that she thought she was not seeing as well as formerly. She was at this time in her third month of pregnancy. The field of vision taken at this time showed marked peripheral contraction but no ring scotoma. The field of vision rapidly narrowed as shown in chart 2.[8]

The important question, naturally, was that concerning pregnancy. In view of the course of the ocular disease, it seemed reasonably certain that some toxin from the gravid uterus was responsible for an exacerbation of retinal disease which

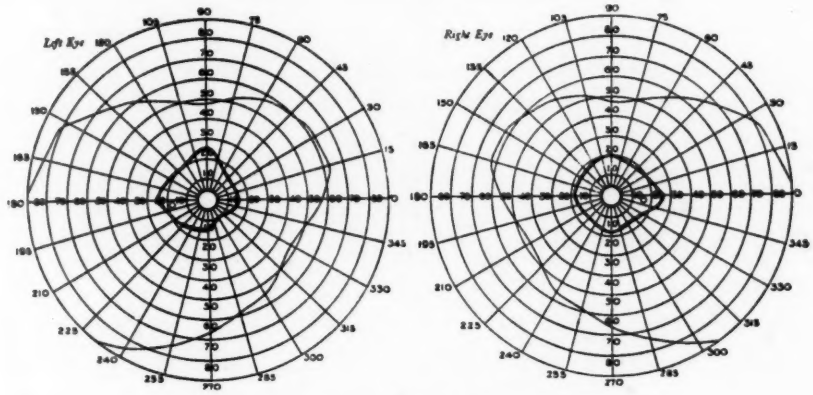

Chart 3.—Visual fields on Sept. 19, 1932.

had lain dormant for almost two years. At no time, however, during the pregnancy did the patient have albuminuria or rising blood pressure and other toxic symptoms.

After consultation, her obstetrician, Dr. Philip H. Smith, decided to interrupt the pregnancy by low cervical cesarean section, which was done on May 2, 1932. Fields taken in September, 1932 (chart 3), and again in January, 1933, showed a slight increase in the form fields. Central vision is still normal; night blindness remains the patient's principal complaint.

The changes in the fundus have already been described, but I wish to emphasize again the marked change which has occurred in the appearance of the fundi during the past few months. The white dots so characteristic of retinitis punctata albescens have, in some portions of the fundi, been replaced by pigmented dots. Over the whole of the retina, there is a marked increase of the deposit of pigment.

8. All fields were taken with daylight illumination on days when the light was of practically uniform brightness. A Schweigger hand perimeter was used with a 5 mm. test object.

SUMMARY

1. Retinitis pigmentosa and retinitis punctata albescens are not frequently found in the same patient.

2. The etiology is unknown. In the case here reported, however, the patient was perfectly well prior to the development of a severe attack of Vincent's angina, following which the retinal disease manifested itself.

3. There was an apparent retrogression of the retinal disease as evidenced by the disappearance of the scotoma and the definite improvement in the hemeralopia.

4. The development of pregnancy apparently precipitated an exacerbation of the retinal disease, evidenced by (*a*) a marked narrowing of the fields, (*b*) an increase in hemeralopia and (*c*) an increase in the amount of pigment.

5. Interruption of the pregnancy at the earliest time compatible with viability of the baby seemed justified in view of the rapid narrowing of the visual fields.

6. Since termination of the pregnancy, there has been no further progression of the defect in the visual fields. There has been some change, as mentioned, in the degree of pigmentation of the retina.

IMPORTANCE OF CEPHALALGIA IN OCULAR DIAGNOSIS

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Ever since Donders pointed out the close connection of optical defects of the eye with cranial pain, innumerable papers and statistics have been published tending to estimate the influence of ocular pathologic processes and of refractive and muscular anomalies on the production of cephalalgia. In other branches of medicine, headache as a diagnostic aid is of limited value. Its presence in the majority of ailments precludes consideration of it as a symptom of a particular disease; hence, most textbooks on general medicine make slight reference to headache. In ocular conditions, however, it is different; cranial pain, as a rule, is stimulated by the use of the eye and subsides when the eye is at rest or when the defect is corrected; consequently, it becomes to the laity and even to some members of the medical profession a symptom pathognomonic of eyestrain.

There is, however, no subject in the entire field of ophthalmology in regard to which there is more divergence of opinion and confusion of figures than ocular headache. There is difference of opinion not only as to the pathologic process but also as to the extent to which the eye is responsible for headache. To give but one illustration, many serious observers claim that as high as 75 per cent of all headaches are ocular in origin; others, equally eminent, particularly European ophthalmologists, scoff at such hyperbolic figures and characterize this opinion as an outgrowth of overspecialization and the thought product of men whose field of vision has become so contracted that they attribute everything to the eyes.

In an attempt to remove some confusion and if possible reconcile divergent views, I present this paper, briefly pointing out the main views on ocular headache in ophthalmologic literature and my own conclusions based on the study of 1,339 patients treated during a period of several years.

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The ancients considered headache a disease;[1] the term cephalalgia was applied to a slight limited or transitory headache; the term cephalea, to severe, deep-seated and chronic pain in the head. They ascribed the cause to some pathologic condition either of the meninges or of some extracranial structure. Galen distinguished between intracranial and extracranial headache; the first, he attributed to irritation of the meninges; the second, to a pathologic condition of tissues outside the cranium. The most important symptom of intracranial headache, according to Galen, is "pain at the root of the eye," "ad radices ocularum." This view was referred to by leading ancient and medieval writers, such as Aetius (sixth century), Paul of Aegina (625-690) and Avicenna (980-1037). The reason given for the intimate relationship of intracranial pain to the eye is that the dura mater, whence headache was thought to arise, forms also the sheath of the optic nerve. Hence, when the dura mater is irritated, pain extends by way of the optic nerve to the root of the eye.[2]

The pathologic process of cranial pain was attributed to certain morbid humors secreted by the brain, itself—considered by ancient anatomists to be a large gland;[3] these humors, when they came in contact with the dura mater, became the irritant, responsible for headache.[4] Hydrotherapy[5] and bloodletting by means of cups applied on both sides of the head were considered the best cure[6] for plethora. Ali ben

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1. The ancient Babylonians, Persians and their Hebrew successors considered headache a serious disease introduced from without by a spirit or specific demon, known in the Talmud as Tsihatho. The demon of hemicrania was known as Palga, from the Aramaic "half." (Talmud Pesahim folia 11b, Sabbath folia 90a, Gittin folia 68b.) This appears to have been the popular belief as late as the sixteenth century. Martin Luther (1483-1546), who believed that demons were responsible for diseases, wrote to his Elector, John of Saxony, when suffering from headache, that "my head is still slightly subject to him who is the enemy of health and of all that is good, he sometimes rides through my brain so that I am not able to read or write."

Some modern writers still consider headache a disease (von Möbius, Paul Julius: Die Migrän, Vienna, Hölder, 1903, p. 104; Wilson, J. C.: Headache Due to General Disease, Symposium on Headache, Proc. Pennsylvania State M. Soc., 1907-1908, p. 14).

2. Campbell, Harry: Headache, and Other Morbid Cephalic Sensations, London, H. K. Lewis, 1894, p. 13.

3. Hirschberg, Julius: Geschichte der Augenheilkunde bei den alten Griechen, in Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1889, vol. 12, chap. 23, p. 68.

4. An old Galenic teaching popular among Arabic physicians up to the fourteenth century.

5. Babylonian Talmud, Sabbath folia 53b.

6. Babylonian Talmud, Gittin folia 68b. Babe Bathra 58b.

Isa [7] (Jesu Haly), the famous Arabian ophthalmologist of the eleventh century, suggested excision or cauterization of the temporal arteries. Bloodletting is still practiced in the Orient and in some parts of eastern Europe. Wet-cups are applied to the back of the neck as a cure or as a preventive of recurrent headache. This practice is usually carried out by the barber of the village at the public bathhouse.

Galen [8] made a curious observation that in sufferers from headache the teeth are apt to be irregular and the palate high. He probably referred to the high palate and Hutchinson's teeth seen in congenital syphilis, associated with headache due to a lesion of the brain.

Next to dysentery, headache was the dread of the Orient. Thus, the Talmud quoted a medical authority, "all sickness I can bear except dysentery, all disease I can overcome except heart disease, and all pain I can tolerate except cranial pain." [9]

A second school of thought, which includes many eighteenth century physicians, stressed the locality of the pain as suggestive of the cause and pathologic condition; as headache is most frequent in the frontal region they believed that the trouble is in the upper air passage or in the membranous lining of the frontal sinus. This opinion appears to have been popular among many ancient Roman and Greek physicians who treated cranial pain by drawing blood from the nose and applying remedies to the mucous membrane of that organ. The famous medieval Arabian physician, Rhazis,[10] wrote that people endowed with a delicate sense of smell are extremely liable to headache. Galen's view, however, found favor among fifteenth and sixteenth century physicians. Thus, Fernelius [11] not only subscribed to the theory of Galen that intracranial pain may affect the eye, but he also believed that even pericranial pain might spread to the eye because of the continuity of the periosteum and other structures with the lining membrane of the orbit.

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7. Hirschberg, J., and Lipperet, J.: Erinnerungsbuch für Augenärzte aus arabischen Handschriften, Leipzig, von Veit, 1904, vol. 3.

8. Kühn, C. G., editor: Medicorum græcorum opera quae extant, Leipzig, 1821-1823, vol 17, p. 815, cited by Harry Campbell. A. Poulard believes that there are ocular headaches of syphilitic origin (Progrès méd. **32**:19 [Jan. 20] 1917). Headache is common in the early stages of acquired syphilis and in hereditary syphilis. The headache of late syphilis is usually symptomatic of arterial change.

9. Babylonian Talmud, Sabbath folia, lla.

10. Rhazis (850-932), famous Arabian physician, known as Ar Razi Mohammed ibn Jakarijah ibn Bekr.

11. Fernelius, A. J. (1497--1558): Medicina, Lutetiæ Parisiorum, apud A. Wechelum, 1554, vol. 2. Fernelius was a French physician, an admirer of the great medical masters of Greece. He gained a reputation for his efforts to revive the study of the old Greek physicians.

The view that pain in the head is extracranial in origin was also supported by the famous anatomist of the seventeenth century, T. Bartholini (1616-1680), who stated that the muscles of the head are the sites of cranial pain.

It is of interest to note that the weight of modern opinion with regard to the causation and pathology of headache is still inclined to the view of the Greek masters. True enough, Galen adhered to the ancient view that headache is a disease and that the ocular involvement accompanying it is secondary to the cranial ailment. There can be no question that he understood the relations of the eye and head, even though the mechanism of reflex headache was not clear to him. A few selected modern views on the etiology and pathogenesis of reflex headache will show how far present day knowledge follows ancient views.

Collins,[12] in line with the old teaching of Bendeletius, considered pain in the head due to extracranial irritation. The occipitofrontalis muscle, he believed, was most liable to pain. Exposure to changes in temperature renders the nerves supporting this muscle liable to compression and subjected to continuous motion. Bendeletius exempted the eye from any responsibility because the latter is well protected by tissues of rich circulation and hence is not subjected to exposure.

Heberden [13] believed that the brain reacts to pain in a manner similar to other organs of the body. On the other hand, Brunton [14] was of the opinion that the changes and constriction are in the extracerebral arteries at their periphery. The blood reaches the constricted part with great impact, causing headache.

Cobb [15] revived the old von Cyon theory that headache is caused by the derangement of the normal circulatory balance, such as a temporary vasodilatation during the rise of systemic arterial pressure, followed by forceful vasoconstriction. Cobb stated that the cerebral vessels contract by stimulation of the cervical sympathetic nerve and dilate when the nerve is cut and when the central end of the vagus is stimulated.

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12. Collins, M.: Dictionnaire encyclopédique des sciences medicales, Paris, V. Masson et fils, 1873.

13. Heberden, W.: Commentaries on the History and Cure of Diseases, ed. 3, London, T. Payne, 1806, p. 92 (cited by Robey, H. W.: Headache, Philadelphia, J. B. Lippincott, 1931, p. 13).

14. Brunton, T. L.: On Disorders of Digestion, Their Consequences and Treatment, New York, The Macmillan Company, 1886, p. 103.

15. Cobb, S.: Cerebral Circulation: Relationship of Cervical Hemorrhage from Venous Capillary Stases, Am. J. M. Sc. **178**:528 (Oct.) 1929. von Cyon, E.: Arch. f. d. ges. Physiol. **74**:97, 1889. Robey, W. H.: Headache, Philadelphia, J. B. Lippincott Company, 1931.

Forbes and Wolff [16] are inclined to the old Heberden theory that cranial pain is due to changes in the functional activities of the vasomotor fibers of the blood vessels of the brain or pia mater.

Weber's [17] explanation of the interrelation between the eye and the cranium is that "the efforts of the ciliary muscles provokes an irregular discharge of nerve energy along the oculomotor nerve to its nucleus, upon which it depends for its proper function. This irritates the neighboring fifth nerve nucleus, with its terminals in the forehead. With this ache, a deep dull pain proceeds from the centers themselves, and from the cortex, through the sympathetic, to the dura mater and soon the whole front of the head aches and a typical eye-strain picture is produced."

Dunn,[18] in a lengthy thesis, endeavored to prove that ocular headache is due both to intracranial and to intra-ocular tension. The increase of tension is physiologic or transitory in contradistinction to the permanent tension of glaucoma.

Clarke [19] did not believe headache to be due to pressure on the dura. He maintained that it is caused by direct vasomotor action on the cerebral and meningeal vessels.

A new theory was that advanced by John Parsons,[20] who expressed the belief that ocular headache is not reflex in character but is protopathic and is influenced directly by the optic thalamus.

Smith [21] was of the opinion that the irritation of functional headaches is extracranial, and gave for his reason that in supra-orbital pain when the maximum tenderness is over the nerve, pressure on the parts will increase the pain.

Tierney,[22] in a comprehensive paper on headache, lauded the old classification, which divides the causes of headache into intracranial and extracranial. Among the extracranial causes, he mentioned diseases of the cranial bones and scalp, and reflex headaches, arising from ocular, nasal, aural and dental disturbances. Dawson [23] endorsed the old galenic

16. Forbes, H. S., and Wolff, H. G.: Cerebral Circulation: III. The Vasomotor Control of Cerebral Vessels, Arch. Neurol. & Psychiat. **19**:1057 (June) 1928.

17. Weber, A.: Eye, Ear, Nose and Throat, Practical Medicine Series, Chicago, Year Book Publishers, 1918, p. 132.

18. Dunn, J. F.: Headache as the Result of Eyestrain, Arch. Ophth. **47**:146, 1918.

19. Clarke, K.: Note on the Common Causes of Persistent Headache and Their Differential Diagnosis, Practitioner **102**:274 (May) 1919.

20. Parsons, J. H.: Diseases of the Eye, ed. 4, London, J. & A. Churchill, 1924, p. 473.

21. Smith, A. B. P.: Lancet **2**:508 (Sept. 8) 1923.

22. Tierney, J. L.: Headache, M. Clin. North America **7**:1515 (March) 1924.

23. Dawson: Some Variety of Headache, Brit. M. J. **1**:607 (April 2) 1927.

theory of tension, explaining that the origin of pain lies in the structures sensitive to pain, which are the dura and its extensions, such as the falx cerebri, the tentorium, the anterior two thirds of the scalp, supplied by the trigeminus nerve, the posterior region of the scalp and the dura of the posterior fossa, which are supplied by the sensory fibers of the upper cervical nerves.

Peritz [24] agreed with Bartholini that functional headache does not arise within the brain centers or the meninges but is induced by myalgias. He added that the severity of a headache depends on the resistance of the nervous system.

Derby [25] expressed the belief that eyestrain is due to, or greatly influenced by, ocular neuroses, which are common, but not frequently recognized by the ophthalmologist.

Michaelson [26] considered that the mechanism of cranial pain is analogous to that which gives rise to reflex visceral pain elsewhere. He substituted the term, "angina capitis" for ocular headache. "Ciliary headache," he said, "arises from ciliary dysfunction, in the same way that angina pectoris arises from cardiac dysfunction."

A new thought based on experimental and clinical studies in line with the suggestion advanced in 1908 by Lewis [27] is that of W. and A. Meerhoff.[28] They ascribed cranial pain to infection by Koch's bacillus. They claimed that there are present with ocular headache, attributed to astigmatism, a host of other apparently insignificant phenomena, among them the symptom complex of hypo-adrenia. These symptoms, they say, disappear in dozens of cases after the administration of tuberculin without any correction by glasses.

In reviewing the various theories proposed to explain ocular cephalalgia, one wonders that none of them offers a satisfactory explanation of the fact that ocular cephalalgia due to eyestrain does not affect alike all persons having the same amount of ametropia. Why does one person suffer intense pain and another, with an equal amount of accommodative or muscular error, have no pain at all? Why is eyestrain manifested by headache in one case and by congestive disturbances in the eye or lid in another?

Much stress has been laid on the nervous make-up of the person, but, while admitting that neurosis may be responsible for cephalalgia in a few cases, I cannot agree that it is the cause in the majority of

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24. Peritz, G.: Zur Pathologie und Therapie der Kopfschmerzen, Med. Klin. **23**:1169 (Aug. 5) 1927.

25. Derby, G. S.: Ocular Neuroses: An Important Cause of So-Called Eyestrain, J. A. M. A. **95**:913 (Sept. 27) 1930.

26. Michaelson, I. C.: Angina Capitis, Brit. J. Ophth. **16**:202 (April) 1932.

27. Lewis, P.: Tr. Sect. Ophth., A. M. A., 1908, p. 59.

28. Meerhoff, W., and Meerhoff, A.: Presse méd. **39**:918 (June 20) 1931.

cases. Of the 805 persons with headache whom I treated, there were many patients evidently in the best of health and entirely free from any nervous disorders. On the other hand, many unmistakably neurotic persons never complained of headache.

A critical analysis of the aforementioned theories and my own studies on the subject lead me to make the following deductions: First, the site of ocular pain is extracranial; it is in the muscles of the head. The dull, bilateral, diffused pain suggests a form of myalgia rather than neuralgia, which is sharp and follows the course of the nerve and its branches rather than its peripheral distribution. It is not the pressure pain characteristic of tumor of the brain and of meningitis. The pain is such that the patient himself often finds it difficult to locate, though he points to certain regions of the head. Second, it reaches the muscles of the head in a reflex manner, analogous to the manner in which reflex visceral pain is caused elsewhere. Third, what plays the most important part in the mechanism responsible for cephalalgia is the presence of an abnormal condition of the nerves in the eye itself, due to abnormalities in its make-up, such as the presence of ametropia and heterophoria.

That the eye is particularly susceptible to neurosis is self-evident, since it is a highly specialized organ. Of the twelve cranial nerves, four go solely to the eye. Of the remaining eight nerves, three have distributions to the eye. It is therefore natural that abnormal structural conditions and defects in the ocular muscle should be associated with abnormalities in the nerve supply. There are, however, instances in which the nerve supply is partially or entirely undisturbed, and in such conditions the patient suffering from eyestrain will experience little pain or may escape cephalalgia altogether. In other words, errors of refraction do not cause headache in persons in whom the ocular nerves are normal; they may produce local symptoms such as congestive disturbance of the eye and lids, fatigue, pain in the visual organ and blurring of sight, but no reflex headache. The severity of headache depends on the degree of functional abnormality of the ocular nerves. The more pronounced the instability of the ocular nerve supply, the more painful is the headache, even though the general nervous condition of the patient is normal.

Among the predisposing causes of ocular headache, heredity is a leading factor. Children inherit from their parents anatomic defects, such as refractive and muscle anomalies. Hereditary transmission of the causes of cranial pain is marked in some families. It may be traced through three or four generations. Howe [29] called attention to the fact that a child's eyes resemble those of its parent or ancestor not only in

29. Howe, Lucien: Note on the Heredity of Corneal Astigmatism and Muscular Anomalies, Tr. Am. Ophth. Soc. **12**:1001, 1911.

the kind of astigmatism but also in the amount and in the meridian of the abnormal corneal curvature. He cites examples of six families in which astigmatism against the rule was a feature of the ametropia and of one family in which the astigmatism of members of two generations was in an oblique diameter. He also noted the same form of heterophoria among members of the family.

Reber,[30] in a study of seven families consisting of 56 persons and covering two generations, found that 34 of 40 patients had exophoria, and in many instances the child had the same kind of headache and neuralgia as the parent.

Atavism sometimes occurs; in large families several members may escape or suffer but rarely from headache. I recall a family of 17 persons, consisting of three generations, in which, with the exception of 2 male members, all were afflicted with cephalalgia. One female member of the second generation escaped, but her children, male and female, were troubled with cephalalgia. The pain among the female members set in about the age of puberty, and among the males several years later.

Transmission is more frequently through the mother to the female children. Not infrequently the paroxysms of a child are almost identical with those of its parent, even to the occurrence of certain other symptoms.

The author found that of a total of 805 patients suffering with ocular headaches, 485, or about 60 per cent, presented a history that one or both parents were troubled with the same condition, and that in 70 per cent of cases the hereditary influence came from the mother.

That the mendelian law of transmission is applicable to the eye has been long recognized by ophthalmologists. Peter [31] was of the opinion that the eye is more influenced by hereditary transmission than any other organ of the body; it is more sharply defined in the eye and not as much obscured by external influences. Nettleship,[32] who has investigated the ocular effect of heredity, gave a long list of transmissible ocular anomalies.

Age is another predisposing cause. Functional headache occurs more frequently in adolescence. Comparatively few children below school age are troubled with headache; headache in this class of cases is due oftener to defective nasal conditions. Ocular headache in children, as a rule, manifests itself after they have attended school a few years. In the presbyopic age, between 40 and 50 years, when rapid failure and even complete loss of accommodation for near vision take place, the

30. Reber, Wendell: Ophth. Rec. **17**:621, 1908.
31. Peter, A.: Lancet **1**:108 (Jan. 13) 1912.
32. Nettleship, E.: Ophthalmic Year Book **9**:398, 1912.

strain of trying to read with inactive ciliary muscles and irresponsive lenses is often the cause of headache. In 100 children under 10 years of age whom I examined, only 15 complained of headache; while in 502 adults between the ages of 21 and 40, 434, or 86.5 per cent, complained of some form of cranial pain. During the presbyopic age, between 40 and 50, the incidence of headache declined to 45 per cent, and in old age there was little headache due to refractive error. The comparative freedom from headache in old age is due to the general blunting of sensation. Errors of refraction in the aged and in very young children are manifested rather by hyperemic symptoms such as blepharitis, conjunctivitis and hordeola (table 1).

Headache is more frequent among women because the percentage of astigmatism is higher among females, particularly those of the young dolichocephalic type. Puberty and menstruation have a tendency to accentuate pain but do not act as a cause of ocular headache. The proportion of men to women with cranial pain is said to range between 30 and 70 per cent, but I did not find quite as low an incidence among men, it being 43 per cent to 57 per cent in women. Of 805 persons with headache, 460 were females and 345 males.

Occupations that entail close use of the eye predispose to cranial pain. To this may be added poor light in stuffy rooms, bad hygienic surroundings, mental strain and fatigue from overwork. Headache is less common among the laboring classes than among merchants and professional men. Patients at the dispensary and in the ward complain less of headache than private patients. The habitual endurance of hardship blunts the sense of pain in the head, and contrariwise the person not used to enduring hard work is more sensitive to pain.

RACE

The Semitic stock and the Celtic and Italic groups appear to possess, on an average, greater sensibility to headache than the Slavonic and Teutonic groups. Primitive races, however, are known to have the same irregularities in the shape and form of the eyes as civilized races of the same land. The difference is in the limited near use of vision by savage people. When primitive man is initiated into civilized life, he is initiated into civilization's symptoms. According to Jones,[33] students at government Indian schools have approximately the same percentage of cranial pain as students at other schools. Of the 64 Negroes in my

33. Jones, C. P.: Study of One Hundred Refraction Cases Among "Indians Fresh from Plains," J. A. M. A. **5**:308 (July 25) 1909. White, J. A., and Pyle, W. L.: Tr. Sect. Ophth., A. M. A., 1908, p. 205.

group of cases, 28, or 45 per cent, complained of cranial pain. The number of Negroes was, however, too small for one to draw any definite conclusions on the basis of color.

A most important factor, often the exciting cause of headache, is impaired health, particularly a broken-down nervous system, following excessive mental activity, anxiety and worry. Neurotic persons are more likely to suffer from headache and associated phenomena, in the presence of an abnormal ocular condition, than those whose nerves are in a healthy state.

Ocular headache may be divided into three classes, in accordance with the abnormal conditions of the eye that produce it: organic diseases, retinal irritation and, the most frequent cause, eyestrain.

The most important organic disease of which headache is a distressing symptom is glaucoma. The pain travels along the branches of the fifth nerve, resembling neuralgia of the trigeminal nerve, and is marked by pressure pain in the temporofrontal region of the affected eye. Cephalalgia, as a rule, is the first symptom of glaucoma. Any severe headache of the temporofrontal region in persons of 40 years or older should cause one to suspect glaucoma.

In iritis, the pain is referred to the orbit, to the temples and along the side of the nose on the affected side. It is worse at night, often so bad that the patient forgets the eye and seeks relief for the cranial pain.

In retrobulbar neuritis, the headache is marked at the top of the head, between the forehead and vertex. It is accompanied by pain in the orbit, often severe and aggravated by movements of, or pressure on, the eyeball.

In choroiditis, cranial pain is occasionally felt at the back of the head. As a general rule, the nearer to the front of the eye the lesion is situated the more is the pain reflected to the surface and the front of the skull; contrariwise, the farther back in the eye the disease is located, the farther back in the head is the headache referred.

In retinal irritation the headache is marked over the brows, radiating to the back of the head. It may be caused by gazing at the sun, as in the observation of a solar eclipse (solar retinitis),[34] by lightning,

34. After the solar eclipse of August, 1932, I saw a case of retinitis in a Negress. The ophthalmoscope revealed the retina to be hyperemic, and there were numerous pigmentary deposits in the macula. The patient complained of photophobia and of metamorphopsia. Her vision was greatly impaired; other symptoms included distention of the superficial and deep conjunctival vessels, swelling of the eyelids and vertical headache. The last was the main reason for which she consulted me.

by bright electric lights,[35] particularly those used in welding, and by exposure to roentgen rays. This type of headache is usually associated with other symptoms, such as conjunctivitis, photophobia, blepharospasm, swelling of the lids, contracted pupils and reduced acuity of vision, due to central scotoma. Retinal cephalalgia may also be caused by gazing at vivid colors, such as colored silks and yarns, and persistent gazing at badly printed or shiny paper.

In dacryocystitis, the pain is usually marked in the orbit of the affected side, and it follows the course of the lacrimal duct.

Headache may occur in cases of albinism with its accompanying symptoms, such as nystagmus and photophobia; also in anisometropia, antimetropia, polycoria and ectopia lentis, because of dazzling and irritation to the visual centers.

EYESTRAIN

The connection of headache with eyestrain was first noted by medical writers in the latter part of the eighteenth century,[36] but the morbid condition responsible for the majority of headaches was described by Donders[37] (1818-1889) of Utrecht, who in 1850 was first to point out that inaccurate optical adjustment of the eyes causes not only diseases of the eye but many other conditions, including headache, nervousness, indigestion, etc. Donders, however, like all persons engaged in pioneer work, did not realize the full clinical importance of his discovery. It was left to S. Weir Mitchel and William Thompson to place the anomalies of refraction on a firm scientific basis—perhaps one of the most important accomplishments in the realm of medicine and surgery. Mitchel,[38] in a series of articles, pointed out that eyestrain

35. In the spring of 1933, I was consulted by an electrician employed at one of the piers in Atlantic City. He presented the history that he tested about 2,000 electric lights within two days; on the afternoon of the second day his vision became blurred and distorted; he saw flashes of light in front of him and a blind spot on looking straight before him, and above all, he had a violent headache extending from the orbits to the vertical region. His pupils were strongly contracted; the retinal vessels were congested, and the retina was hazy; there was defective color vision but no conjunctival or corneal involvement. The patient fully recovered in two weeks.

36. The symptom of "weak eyes" associated with pain in the eyebrow was described by Taylor (1776), Plenck (1792), Scarpa (1816) and Beer (1817); later by Lawrence, Tyrrel, Sichel, and MacKenzie. Risley, S. D., in Posey, William, and Spiller, W. G.: The Eye and the Nervous System, Philadelphia, J. B. Lippincott Company, 1906, p. 744.

37. Donders, F. C.: Anomalies of Refraction and Accommodation, London, New Sydenham Society, 1864.

38. Mitchel, S. W.: Headaches from Heat-Strokes, from Fevers after Meningitis, from Overuse of the Brain, from Eye-Strain, M. & S. Reporter **32**:67, 1874; Notes on Headaches, ibid. **32**:101, 1875; Headaches from Eye Strain, Am. J. M. Sc. **71**:363 (April) 1876.

may manifest itself only by frontal or occipital headache, without any pain or even fatigue in the eyes.

About three years later (1879), Thompson [39] published a lecture he had delivered before the students of Jefferson Medical College, entitled "Astigmatism as a Cause of Persistent Headache and Other Nervous Symptoms." He became aware for more than ten years, he stated in the publication, "that patients who consulted him for defective vision presented symptoms of pain in the eyes, brows, temples and occiput" and a series of other nervous manifestation. He called the attention of S. Weir Mitchel to this phenomenon; the latter, being convinced of the importance of the discovery, impressed the facts on the medical world.

In England, Thomas Young was first to demonstrate astigmatism in its relation to cephalalgia and other nervous phenomena. Another Englishman, Sir George Airy, the astronomer, described astigmatism in his own case—it was a case of anisometropia in which one eye was affected with compound myopic astigmatism.[40] His son, Dr. Hubert Airy, from the description he gave, appears to have also suffered from headache due to astigmatism.[41] His attacks came on after close work; he noticed parts of the words were dimmed by clouded spots.

George M. Gould, in his characteristic, forceful way, sought to prove the relation of eyestrain to morbid psychology among literary worthies. In a most interesting volume entitled, "Biographic Clinics," he stated that the continuous ill health of De Quincey, the Carlyles, Darwin, Huxley, George Eliot, Browning and many others was the result of eyestrain. He endeavored to prove that all persons whose histories he describes suffered because of near use of the eye and that they obtained relief only when they put reading or writing aside and took long walks or horseback exercise in the country, but when they resumed literary and scientific work, the headache, insomnia, irritability of temper and depression of spirit returned.[42]

Several years later (1884), Stevens,[43] in an analysis of a large number of patients, based on eleven years of observation, also reached the concluson that cranial pain and many other neuropathologic conditions are the result of the injurious effects of muscle imbalance on

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39. Thompson, William: M. News & Libr. **37**:81 (June) 1879; in Garrison, F. H.: History of Medicine, Philadelphia, W. B. Saunders Company, 1913, p. 578.

40. Airy, George: Philosoph. Mag. **30**:19 (July) 1865.

41. Airy, Hubert: Tr. Roy. Soc. London, 1870, p. 16.

42. Gould, G. M.: The Ill Health of Richard Wagner, Lancet **2**:306 (Aug. 1) 1903; The Rôle of Eye-Strain in Civilization, Brit. M. J. **2**:663 and 775, 1903.

43. Stevens, G. F.: Functional Nervous Diseases, New York, D. Appleton and Company, 1887.

the eye and nervous system. His practical applications to the correction of muscular anomalies made an important contribution to the science of ophthalmology.

The result of these investigations is now recognized by both physician and layman. Both turn to the oculist in cases of obstinate headache, and frequently the patient consults the oculist of his own accord, basing his own diagnosis of ocular trouble on the presence of cranial pain.

The percentage of cephalalgia due to ocular causes given by different observers [44] ranges from 30 to 80; both figures are probably extremes; a middle course of 50 per cent would be nearer the truth. The estimates of de Schweinitz [45] that fully 60 per cent of all functional headaches are due to ocular strain and of Wood [46] that 75 per cent of all bilateral frontal headache is ocular in origin have a basis of fact. The conflict of figures arises from the fact that the frequency of a common symptom, such as headache, is difficult to estimate. Practitioners and specialists may not record pain in the head due to causes remote from the province of their practice.

Some of the statistical data published in ophthalmologic literature may throw light on the percentage of headaches among ophthalmic patients. Zimmerman,[47] in an analysis of 2,000 ophthalmic patients, found that 71 per cent suffered from pain in the head; the same figures were reached by Snell [48] from 1,010 patients. Risley,[49] in the study of 1,000 ophthalmic patients, found that 50 per cent had ocular cephalalgia. Walton [50] found one half of all headaches to be caused by eyestrain. Wood expressed the belief that the ocular element in all forms of headache is probably not less than 40 per cent, but Snydacker,[51] who found headache in 40 per cent of 2,000 patients, questions its ocular

44. Hansel, H., in Posey, William, and Spiller, W. G.: The Eye and the Nervous System, Philadelphia, J. B. Lippincott Company, 1903, p. 888.

45. de Schweinitz, G. E.: Diseases of the Eye, Philadelphia, W. B. Saunders Company, 1906, p. 180.

46. Wood, Casey: System of Ophthalmic Therapeutics, Chicago, Cleveland Press, 1909, p. 827.

47. Zimmerman, M. W.: Ocular and Other Reflexes, New York & Philadelphia M. J. **78**:973 (Nov. 21) 1903.

48. Snell, A. C.: The Relation of Headache to Functional Monocularity, Arch. Ophth. **51**:5, 1922.

49. Risley, S. D., in Posey, William, and Spiller, W. G.: The Eye and the Nervous System, Philadelphia, J. B. Lippincott Company, 1903, p. 747.

50. Walton, G. L.: Eye Strain: Its Importance and Its Limitations, Boston M. & S. J. **152**:713 (June 22) 1905.

51. Snydacker, E. F.: Zur Differentialdiagnose zwischen okularen Kopfschmerzen und solchen, verursacht von Nebenhöhlenentzündungen, Klin. Monatsbl. f. Augenh. **47**:629 (July) 1909.

origin. Mayer,[52] a neurologist, maintained that 50 per cent of the headaches are in patients referred by the family physician, who has eliminated all other causes.

In the analysis of 1,339 ophthalmic patients of all social types, I found 805, or 60 per cent, complaining of headache. Of these, 86 per cent were due to asthenopia and the rest to other conditions such as iritis, glaucoma, retinal irritation, uveitis, choroiditis and congestive disturbances of the lids, conjunctiva or lacrimal apparatus. Of course, the accuracy of all statistics depends on the type of one's practice. In a large industrial practice one sees fewer cases of headache than in a clientele of literary or professional men.

In some European countries there is a tendency to minimize the ocular origin of cephalalgia. The average person does not consult the oculist about his headache. The use of spectacles among the lower classes is limited. Charles [53] expressed the belief that the American nervous system in contrast to that of other nationalities is more sensitive to pain, and a very low grade of astigmatism requires correction even among the lower classes. Charles tells of a case in which he did the refraction in the presence of Professor Voelker of Kiel; he took much pain to uncover an error requiring a + 3.00 sphere combined with a + 3.00 cylinder. The professor evidently considered it a waste of time, as he remarked, "You need not bother about the astigmatism, these stupid people cannot make such distinctions." Be it as it may, European oculists do not hold the eye responsible for much of the cranial pain. Maitland Ramsay [54] does not agree that there is a large percentage of headache of ocular origin as claimed by American oculists. Gunning went a step further when he stated that errors of refraction are responsible for few cases of cranial pain. Myopia, he said, "did not cause head-pain except when over corrected and astigmatism only when in oblique diameter, seldom when the axis is vertical or horizontal and even in these, there are cases where the headache will not disappear with full correction."

Of recent years this tendency to underestimate ocular causes in the production of headache is not confined to the other side of the Atlantic. Charles [53] pointed out that the eye may be only the exciting cause of headache; errors of refraction by themselves may not be strong enough to produce cranial pain if the patient has a well balanced nervous system. Walton [55] holds a similar view. "The alleviation of headache

52. Mayer, E.: Headache Due to Nervous Diseases, in Symposium on Headache, Pennsylvania M. J., 1907, vol. 9.

53. Charles, J. W.: Ocular Headache, J. A. M. A. **71**:1711 (Nov. 23) 1918.

54. Ramsay, A. M.: Clinical Ophthalmology for the General Practitioner. New York, Oxford University Press, 1920, p. 16.

55. Walton, G. L.: Migrain and Occupation Neuroses, Tr. Sect. Ophth. A. M. A., 1908, p. 425.

by glasses is an indication that the eyes are sometimes the exciting or aggravating cause."

Tucker,[56] with twenty years of observation of a large number of patients, concluded that headache due to refractive or muscular errors is not nearly as common as is generally believed. He asserted that there are a group of neurasthenic persons who are relieved by the suggestive effect of wearing glasses. Coggeshall and MacCoy [57] stated that neurasthenic diathesis is an essential underlying condition in the occurrence of chronic headaches. Gradle [58] expressed the belief that the difference in the symptoms depends on the condition of the nervous system. Errors of refraction do not cause headache in a normal person. They cause only local discomfort, such as congestive disturbance of the eye or its appendages.

Derby,[59] in a comprehensive paper, "Ocular Neuroses," suggested the application of psychoneurologic treatment for eyestrain to bring the patient to a subconscious level in order to offset eye-consciousness. He was of the opinion that commonsense suggestions are all that is needed in 95 per cent of cases, and a complete history of the case will be of much value.

Rutherford [60] differentiated between asthenopia and eyestrain; the first is a visual discomfort that attends prolonged close work and is accompanied by fatigue and reflex disturbance remote from the eye, while eyestrain is limited to the eye itself and disappears as soon as prolonged use of the eye stops. He identified asthenopia as a psychoneurosis and advised that it should be treated as such.

It is not my purpose in this paper to detract from the importance of the nervous element associated with pain anywhere, but to remove ocular headache from the legitimate place established by Donders and Mitchel and to place it in the province of the psychoneurologist is against the every-day experience of the ophthalmologist, who bears witness to the complete relief afforded by the correction of accommodative and muscular errors in a large majority of patients. Even though the relation between ocular defect and pain in the head is not established by

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56. Tucker, J. H.: Headache Due to Ocular Disease, South. M. & S. J. **92**:71 (Feb.) 1930.

57. Coggeshall, Frederick, and MacCoy, W. E.: Headache as a Symptom of Local Disorders, J. A. M. A. **50**:15 (Jan. 4) 1908.

58. Gradle, H.: Etiologic Studies of Neurosis of Peripheral Origin, J. A. M. A. **34**:1166 (July 12) 1900.

59. Derby, G. S.: Ocular Neuroses, J. A. M. A. **95**:913 (Sept. 27) 1930; Tr. Sect. Ophth., A. M. A., 1930, p. 305.

60. Rutherford, C. W.: Asthenopia, a Psychoneurosis and a Medical Responsibility, J. A. M. A. **99**:284 (July 23) 1932.

an oculist—as when correction of anomalies affords no relief—this is no argument against the ocular origin of the pain, as it often happens that another, more exacting oculist may detect a slight error.

To make neurosis the predominating factor in ocular headache is to substitute the unknown for the known. Ametropia and heterophoria are demonstrable phenomena that can be measured; a sensitive, unstable nervous system cannot be so easily demonstrated. While the general condition of health and an unbalanced nervous system might be regarded as a contributing cause, the primary factors in cephalalgia are anomalies in the eye and muscles. Treatment should be directed to correcting these defects. Psychoneurosis, like focal infection and endocrine imbalance, cannot be made the cause of all human aches. These conditions have their reserved places.

Ocular headache may be divided into "bilateral-symmetrical" (pain of equal intensity on both sides) and "bilateral-asymmetrical" (pain more severe on one side). The bilateral-symmetrical is the most common form of ocular cephalalgia. One, however, meets with the asymmetrical type when one eye has a greater optical defect than the other; for example, when one eye is astigmatic and the other is simply hypermetropic, or when one eye is astigmatic with the rule and the other eye against the rule. Oliver [61] pointed out that hypermetropic astigmatism against the rule is more apt to produce pain in the head than the same amount and form of astigmatism with the rule, and oblique meridians are more likely to give rise to headache than the vertical and the horizontal meridians. One fourth of a diopter of astigmatism in an oblique direction may cause much distress.

Ocular pain seldom occupies the entire surface of the cranium. It is generally limited to a certain region or regions. The seat of pain also changes at times. Persons susceptible to headache by no means suffer at the same place all the time.

Ocular headache differs from the cranial pain of other causes in the mode of onset. It usually begins with a sensation of heaviness in the head, followed by a dull pain which gradually becomes more acute and localized in one or more regions of the head. It is described, as to degree, as acute, sharp, dull, slight, severe, transitory, constant, superficial and deep-seated; as to character, as splitting, throbbing, stabbing, burning, crossing, pressing and cutting. It is usually located with sufficient definiteness in a certain part of the head, so that one speaks of it as frontal, occipital, temporal, orbital or parietal. Neither the degree nor the character of the pain is of much value in ocular diagnosis.

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61. Oliver, C. A., in Norris, W. F., and Oliver, C. A.: System of Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1900, vol. 4, p. 449.

There is, however, a difference of opinion as to the location, as a writer in the *Glasgow Medical Journal* pointed out. "The synapse guarding the brain tracts of the ophthalmic division of the trigeminus appears to provide an outlet for impulses able to overflow. The greatest majority of headaches are frontal, but the location of any headache is not an indication of the nature and source of the disturbing impulse." [62] On the other hand, Lauder Brunton [63] and others expressed the belief that the location is suggestive of the source of cranial pain; thus it is accepted by many writers that supra-orbital and frontal headache indicates hypermetropia; [64] occipital pain points to muscular insufficiency,[65] and temporal pain is a sign of hypermetropic or myopic astigmatism.[66] Iritis and early glaucoma are often marked by distressing pain in the orbits; postocular neuritis and diseases of the optic nerve frequently manifest themselves by pain in the vertex.

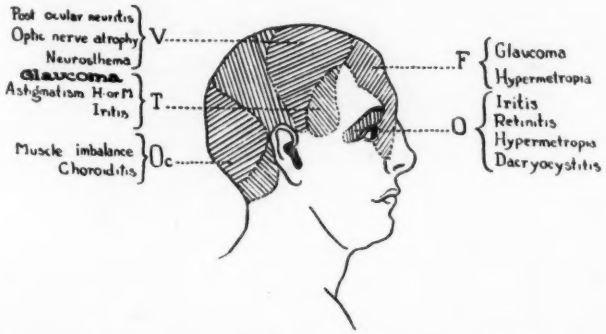

Fig. 1.—The areas where cephalalgia is most frequent in various ocular conditions or diseases. *V* indicates vertical; *T*, temporal; *Oc*, occipital; *F*, frontal; *O*, orbital.

As to frequency, Wood gives the following order: supra-orbital, deep orbital, frontal, occipital and temporal. Zimmerman,[47] in his statis-

62. Cited in Eye, Ear, Nose and Throat, Practical Medicine Series, Chicago, Year Book Publishers, 1917, p. 119.

63. "It would be going too far to say," stated Brunton, "that frontal headache is always due to an abnormal eye condition but I believe that it is more frequent than one would suspect." St. Bartholomew's Hosp. Rep. **19**:336, 1883.

64. Fuchs, E.: Text Book of Ophthalmology, ed. 3, translated by A. Duane, Philadelphia, J. B. Lippincott Company, 1908. Risley, S. D.: Relation of Headache to Affections of the Eye, Pennsylvania M. J. **11**:179, 1907. Munson, J. H.: Headache, Philadelphia, Lea & Febiger, 1913, p. 46. Kierney, J. H.: Ocular Factor in Headache, New York M. J. **114**:565, 1921. "Headache," Cyclopedia of Medicine, edited by George M. Piersol, Philadelphia, F. A. Davis Company, 1932.

65. de Schweinitz,[45] p. 696.

66. Reilly, Thomas: Headache, Philadelphia, P. Blakiston's Sons & Company, 1926, p. 135.

tical study of 2,000 cases of headache, found 71 per cent in the frontal region, 16 per cent in the fronto-occipital region and the remaining 14 per cent divided among the temporal, occipital and vertical regions. He does not believe muscle imbalance to be a common cause of cranial pain. I found muscular anomalies in over 20 per cent of cases of cephalalgia. I also ascertained that the site of ophthalmic headache varies in frequency in the following order: supra-orbital and frontal, 52 per cent; temporal, 15 per cent; occipital, 11 per cent; parietal or vertical, 8 per cent; unilateral, 5 per cent, and general, 10 per cent. General headache, as a rule, is due to the inaccuracy of localization of pain

TABLE 1.—*Classifications of Asthenopic Symptoms According to Age of Patients*

	Age of Patients							
	6-10	10-15	15-20	20-40	40-Over	Total	Male	Female
Number of patients	100	107	115	502	515	1,339	629	710
Number of refractions	70	64	99	470	442	1,145	509	636
Number of patients with headache	15	42	82	434	232	805	345	460
Areas where headache is most frequent								
Frontal pain	17	15	27	119	101	279		
Orbital pain	6	5	13	85	26	135		
Temporal pain	3	5	14	60	38	120		
Occipital pain	2	3	6	55	19	85		
Parietal pain	1	0	2	36	23	62		
General pain	4	7	13	45	14	83		
Unilateral pain	0	1	3	30	7	41		
Hyperemic symptoms without headache	13	22	28	48	14	127		
Ocular pain or fatigue with or without headache	6	16	26	184	129	361		
Blurred vision with or without headache	2	9	46	223	243	523		
Reflex neurosis of various types	15	10	15	15	10	60		
Monocular convergent squint	16	5	3	19	29	72		
Binocular convergent squint	3	0	1	13	8	25		
Monocular divergent squint.	0	0	1	22	17	40		
Binocular divergent squint	0	0	0	11	4	15		
Hereditary	..	..	..	..	..	485	145	340

by the patient. Ocular cephalalgia seldom affects the entire head. At times pain in the head involves more than one, not necessarily neighboring, area. Hence one speaks of fronto-occipital, frontotemporal and frontoparietal headaches. The first frequently signifies hypermetropia combined with lateral heterophoria; the second points to hypermetropia with astigmatism, and the last suggests hypermetropia associated with some organic trouble of the eye.

Ophthalmic headache caused by excessive work placed on the muscles of accommodation and fixation follows the continuous use of the eye and comes on with such regularity that the patient himself connects the pain with the use of the eyes. Ocular pain, as a rule, is at its height in the late afternoon hours and at its decline in the morning; occasionally, however, one meets with a patient who complains of headache after a night of rest. The pain may follow a sound sleep after a busy

week or after an evening of reading. True, during sleep the mechanisms of accommodation and of fixation are at rest, and the strain of the ciliary and ocular muscles is relieved, but with the opening of the eyes the strain on the overworked muscles is suddenly reestablished, and the abrupt contraction of the ciliary muscles in this case is a greater factor in the causation of headache than the amount of contraction demanded when a slower adaptation takes place. Ocular pain is intensified when the pupils are contracted as when one is reading before a bright light or when myotic drugs are used, and it is particularly severe in occupations requiring rapid changes in focus, such as that of pianists and typists before the necessary movements of the fingers have become sufficiently automatic not to require gazing at the keyboard; injurious as such occupations are at any time, they are doubly so when they are carried on in bad light.

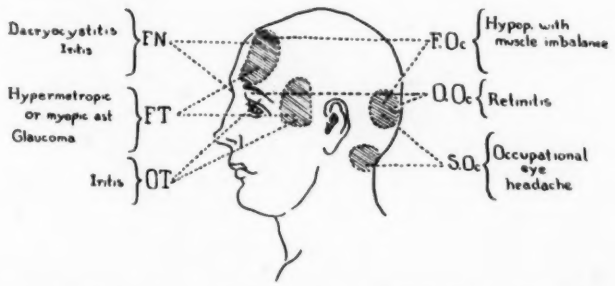

Fig. 2.—The regions where cephalalgia is present when more than one area is involved. *FN* indicates frontonasal; *FT*, frontotemporal; *O.Oc*, orbito-occipital; *OT*, orbitotemporal; *F.Oc*, fronto-occipital; *S.Oc*, suboccipital.

The factors responsible for headaches are first hypermetropia; the short-eyed person, in order to focus near objects, must bring the power of accommodation into play; the constant overaction of the ciliary muscles controlling the act of accommodation produces fatigue and spasm, followed by headache. Second in responsibility for ocular pain is astigmatism—hypermetropic, myopic or mixed. In the irregularly shaped eye, cranial pain may be the result, even on fixation at a distance. The factor in producing the strain is due to the continuous alteration of accommodation, which takes place in order that first one meridian of an object and then the other may be brought into focus. If the vertical view is focused, the horizontal view is dim and vice versa. This continuous change of accommodation is tiring to the ciliary muscles.

Snell,[67] in an analysis of 300 cases, found the following forms of ametropia in the order here given: simple and compound hyper-

67. Snell, Simon: Eye-Strain as a Cause of Headache, London, Simpkin, Marshall, Hamilton Kent Co., 1904, p. 34.

TABLE 2.—*The Degree of Ametropia According to Age of Patients Complaining of Headache and Other Ocular Symptoms* *

Condition		6-10	10-15	15-20	20-40	40-Over	Total Number		
	+								
Hypermetropia.....	0.50 D.	4	4	2	12	10	32		
	1.00	0	0	5	18	16	39		
	1.50	3	1	2	11	9	26		
	2.00	1	0	1	9	14	25		
	Over	0	2	0	13	13	28		
							——	150	(14%)
	+								
Hypermetropic astigmatism	0.50 D.	2	1	6	18	12	39		
	1.00	0	0	1	10	12	23		
	1.50	0	1	0	7	5	13		
	2.00	0	1	1	3	3	8		
	Over	1	0	1	7	8	17		
							——	100	(9%)
	+								
Compound hypermetropic astigmatism	0.50 D.	3	2	2	16	9	32		
	1.00	5	6	6	24	20	61		
	1.50	5	2	2	23	28	60		
	2.00	5	2	2	21	22	52		
	Over	12	12	7	42	46	118		
							——	323	(28%)
	−								
Myopia.....	0.50 D.	1	0	3	3	7	14		
	1.00	1	1	4	7	11	24		
	1.50	1	0	1	3	12	17		
	2.00	0	2	0	..	6	8		
	Over	1	6	2	..	16	25		
							——	88	(8%)
	−								
Myopic astigmatism	0.50 D.	0	0	0	12	10	22		
	1.00	1	0	1	5	2	9		
	1.50	0	0	0	8	14	22		
	2.00	0	0	0	12	5	17		
	Over	0	0	3	6	12	21		
							——	91	(8%)
	−								
Compound myopic astigmatism	0.50 D.	1	0	2	24	4	31		
	1.00	4	5	4	24	15	52		
	1.50	1	5	3	25	12	46		
	2.00	5	4	6	23	6	44		
	Over	7	5	18	33	36	99		
							——	272	(24%)
Mixed astigmatism	±	6	4	14	50	47	121		(11%)
Total.....		70	66	99	468	442	1,145		(80% of 1,339)

* The analysis presented here is based on the study of 1,339 persons treated during a period of several years. The ocular life of the patients has been divided into five periods: first, up to 10 years of age; second, from 10 to 15; third, from 15 to 20; fourth, from 20 to 40; and fifth, over 40 years of age. These ages present definite periods and have different demands on the eyes. The corrections of ametropia as arranged in this table are in halves and units; 0.50 D. stands for lenses of 0.50 D. and less in either or both eyes; 1.00 D. for 1.00 D. and less, e. g., 0.62 and 0.75 D., etc.

As ocular headaches are due mainly to errors of the refractive apparatus, it was thought advisable to tabulate the headaches in accordance with the strength of the lens that neutralized the error of refraction and incidentally relieved the headache. Of the 1,339 patients treated, 805, or about 60 per cent, complained of headache, and of 1,145 persons with errors of refraction about 70 per cent were troubled with cranial pain. The errors in 376 cases were neutralized by lenses of 0.25, 0.50 or 1.00 D. sph. or spherocylinder; 177, by 1.50 D.; 149, by 2.00 D. Only 112 patients required a stronger lens than 2.00 D.

metropic astigmatism, 49.3 per cent; hypermetropia, 34.3 per cent; simple and compound myopic astigmatism, 11.3 per cent; mixed astigmatism, 3.6 per cent, and myopia, 1.3 per cent. From my 1,145 cases in which refraction was done I compiled the following statistics: compound hypermetropic astigmatism, 28 per cent; compound myopic astigmatism, 24 per cent; hypermetropia, 14 per cent; mixed astigmatism, 11 per cent; simple hypermetropic astigmatism, 9 per cent; simple myopic astigmatism, 8 per cent, and simple myopia, 8 per cent. The short eye of the person with hypermetropia and the irregularly formed astigmatic cornea of both varieties are responsible for the majority of ocular headaches.

Next in importance and contributing about 20 per cent to the cases of pain in the head is muscle imbalance, producing the phorias and especially those of the vertical variety. The pain is most common in the occipital region; it may follow the use of the eye immediately or be delayed and come on at any time of the day. It is probable that when the pain is associated with nausea, dizziness and vertigo the extraocular muscles are at fault.

Suboccipital headache is frequent among patients whose occupations demand constant elevation of the eyes and frequent changes of fixation. These include carpenters, painters and paperhangers, in whom there is in addition a strain on the accessory muscles of the eye, such as the recti capitis and the scaleni, the consequence of which is pain in the lower occipital region extending down the neck, with tender points corresponding to the attachment of the occipital muscle. The correction of the ocular defects may relieve the pain in the occiput and ease the tenderness at the base of the head. The headache due to occupation may linger in a mild form after ocular anomalies have been rectified because the patient has formed a headache habit.

Pain at the base of the skull, radiating from the frontal region, is said to signify hypermetropia combined with lateral heterophoria. Symptoms appear after an evening in a motion picture theater, an afternoon in the shopping districts or a train ride. Continuous changes in fixation at these times impose excessive demands on the muscles of the eye. Peter [68] used the term panoramic headache for those caused by muscular asthenia; he said that panoramic headache is aggravated by close work and increases as the day advances. A night's rest brings relief.

Accommodative and muscular anomalies do not always cause headache. A congenital defect may exist for a long time without causing

68. Peter, L. C.: The Extra-Ocular Muscles, Philadelphia, Lea & Febiger, 1927, p. 121. (Van der Brugh found esophoria in a special group of persons who came home with headache from theaters, movies, lecture halls, concerts or churches. A special feature in these cases is a tendency for the pain to subside in a dark room or at night.)

any discomfort. Symptoms appear only after a breakdown of the system, as, for example, after a surgical operation or a prolonged illness. Every oculist has discovered in a person with presbyopia coming primarily for reading glasses, refractive errors of high degree, such as hypermetropia and astigmatism or a heterophoria which had been no handicap in school, business or professional life. Other patients have suffered all sorts of discomforts from small abnormalities. Indeed, it is the unsuspected low degree of ametropia and heterophoria with normal vision or better vision that causes great reflex disturbances. In extreme errors, the anomaly cannot be overcome; the muscles abandon the effort. The patient submits to the inevitable and makes no effort to see, and strain is at an end. Thus, in chidren amblyopia exanopsia and strabismus develop. With a low error, the patient tries to overcome the deficiency. Overaction and hypertension of the muscles result in asthenopia and headache. The cranial pain in these cases is of diagnostic value. Because of the good vision, the eyes are not suspected. Relief often follows the use of a low sphere or spherocylinder which may blur slightly at a distance. The glass is to be worn notwithstanding that fact.

The majority of patients with ocular headache have good distant vision.[46] In the 1,339 cases analyzed in this paper, headaches of greatest severity occurred in patients with extra good vision, most of whom required a small correction. In 805 patients with headache the 367 who suffered worst were relieved by wearing a lens ranging from 0.25, 0.50 to 1.00 D. sphere or sphero-cylinder of either sign. One hundred and seventy-seven persons were relieved by 1.50 D. and 149 by a 2.00 D. lens. Only 112 patients required a lens stronger than 2.00 D (table 2).

There are instances, however, in many cases of high hyperopic astigmatism when the patient tries to overcome a blur by contracting the orbital fissure to form a sort of stenopeic slit. This causes supraorbital pain residing in the orbicularis palpebrarum. The same kind of pain is experienced by those who contract these muscles as a protection against the sun.

In anisometropia and antimetropia, cranial pain is due primarily to the constant strife between two ocular muscles. Another factor is the marked difference in the size of the retinal images, causing a conflict in fusion.

The cephalalgia occasionally met with in simple myopia is due to stretching of the choroid and sclera, and is found in the progressive type, in which case the pain is in the back of the head. In simple myopia pain in the head of a reflex phenomena is unusual. Myopic patients are more likely to be troubled with hyperemia of the conjunctiva and lids and pain in the eyeballs, due to overwork of the internal recti muscles.

According to Risley,[69] patients with ocular headache rarely have congestive disturbances, such as red lids, inflamed conjunctiva and recurrent styes. On the other hand, patients troubled with inflammatory symptoms as a result of asthenopia do not, as a rule, complain of headache. I found about 15 per cent of asthenopic patients with exclusively inflammatory symptoms. Cephalalgic and congestive symptoms do not occur simultaneously. Local symptoms, however, may follow headache in rare cases (table 1).

Ocular headache is infrequent among persons with vision restricted to one eye, as result of injury or disease. Cranial pain is rare in cases of greatly impaired vision. The frequency and severity of headache is in inverse ratio to the visual acuity. The better the vision, the more headache occurs; the poorer the vision, the less headache. This phenomenon may be expressed in the familiar saying, "What you don't see doesn't hurt you."

Ophthalmologists have long observed that patients with monocular vision suffer little from cephalagia. Donders [70] observed that asthenopia is rare among persons with strabismus. Oliver [61] remarked that if mankind were monocular the problem of asthenopia would not exist. Snell,[48] in an exhaustive study of the relation of headache to functional monocularity, proved conclusively that there is a "smaller percentage of headache, in proportion to the greater degree, or completeness of monocularity." The reason is that in cases in which vision is centered in one eye the complexity of the fusion sense is eliminated, and the muscular adjustment necessary to maintain single binocular vision needs no coordination; the visual act, therefore, is under less nervous strain.

While no particular type of pain in the head is pathognomonic of asthenopia, the fact that the majority of ocular cephalalgias are frontal is suggestive. If the frontal headache is low and seems to emerge from the eyes and if it comes on during or after the prolonged use of the eyes at close work, it is more than suggestive. Students, bookkeepers, writers and persons with similar occupations who rest on Sunday experience relief on that day if the headache is of ocular origin. Manual workers who indulge in much reading over the week-end suffer only then. If a complete rest to the eyes give relief or if a mydriatic is employed and relief is experienced, one is quite sure of the etiology. Extrinsic muscle imbalance may be suspected if the patient discovers that there is no fatigue when only one eye is used, the other being covered, or when the patient is annoyed by a tendency toward diplopia

69. Risley, S. D.: Symposium on Headache, Proc. Pennsylvania State M. Soc., 1907, p. 7.

70. Donders, F. C.: Anomalies of Refraction and Accommodation, London, New Sydenham Society, 1864, p. 415.

when looking at a distance (owing to the fact that when the accommodation is relaxed, the abnormal muscular effort required to maintain binocular vision is also relaxed, thus manifesting a latent deviation).

Van der Brugh's [71] test may be tried in such cases. This consists of the closing of one eye for three days; if the headache stops, it was probably due to loss of balance and may be corrected by prism practice or prismatic glasses.

There are, however, cases in which the connection of headache with the eye cannot be easily traced; the cranial pain is continuous and not aggravated by the use of the eyes. The retinoscope and trial case do not reveal any errors of refraction, yet the fluffy retina, the hyperemic nerve head and other ocular manifestations are symptoms of strain.

There is another and by no means an inconsiderable group of patients, who have absolutely no abnormalities, on whom the wearing of glasses has a psychic effect like that of the wearing of amulets or religious charms. This group of patients is composed mostly of women whose reproductive organs have been removed.[69] There are still other cases of cephalalgia that are unquestionably due to several contributory causes—those in which asthenopia is only one factor. In cases of this type the pain will not disappear altogether following optical treatment.

A safe practice is to consider all headaches ocular in origin until proved otherwise. The proof to consist not only of firmly fixing the other pathologic condition responsible for the pain, but also the positive elimination of ocular trouble. The differential diagnosis between headache due to organic diseases and ocular cephalalgia can readily be made.

Cephalalgia due to organic diseases is characterized by its persistency; it sometimes undergoes exacerbation and is aggravated by physical efforts, but is rarely wholly absent. It has a tendency to increase in severity and interferes with restful sleep. Ocular cephalalgia is not persistent; it is not, as a rule, made worse by physical toil and is absent when the eyes are at rest.

The most important varieties of headache which possess somewhat distinctive characteristics are as follows: Nephritic headache—except the sudden attacks due to uremia—is in most cases caused by the arteriosclerosis which so often forms an essential part of chronic nephritis. The pain is apt to be of a throbbing character, somewhat shifting and often accompanied by vertigo and tinnitus. Headache from disorders of digestion and constipation is usually of a throbbing, pulsating character; it affects the frontal and orbital regions, and is made worse by sudden movements of the head. The headache of uterine diseases is usually occipital, sharp and radiating. Anemic headache is a sore

71. Van der Brugh, J. P.: Chronic Headache, Nederl. tijdschr. v. geneesk. 2:132 (July 14) 1917.

and pressing pain, usually felt in the forehead and orbital region or in the vertex, and is often associated with occipital pressure. As its name indicates, it is found in connection with the general and special forms of impoverished blood.

In the typical headache of hysteria the patient feels as if a nail was being driven into the top of the head (clavus), but this type is of comparatively infrequent occurrence. The headache of neurasthenia, probably the most frequent of all headaches which require treatment, is of a pressing character; it is usually vertical, but is sometimes described as a band around the head. It is almost invariably worse in the morning, becoming lighter or disappearing toward the latter part of the day. Headache from turbinal pressure is felt as a pain beginning at the root of the nose and running directly backward to the occiput. It is greatly increased in severity by coughing or bending over. In chronic nasal trouble the headache continues during the night, is accompanied by a discharge from the nose and is aggravated by an attack of coryza. The headache due to a pathologic condition of the frontal sinus is marked by pain in the upper and inner part of the orbit, often on one side only; there is tenderness on pressure on the orbital roof. The headache is present in the morning and, owing to better drainage when the patient is in an upright position, it passes off at midday with a slight flow of mucous discharge.[72] Ocular headache is not affected by simple coryza, is not accompanied by discharge from the nose and is not present when the patient retires. When there is no strain there is no headache.

The cephalalgia due to migraine, particularly that of the ophthalmic type, is distinguished from the pure headache of eyestrain in its consistent similarity and in the periodicity of its attacks. It is frequently characterized by an aura and distorted colored vision. The pain is rapid in development, is usually located in the temporofrontal region of one side of the head and is often followed by nausea and vomiting. The attacks usually begin at puberty and cease between the fortieth and fiftieth years of life; the pain lasts from six to twenty-four hours and completely disappears. An ocular examination should be made of all patients suffering from migraine, for almost all have ocular defects—generally hypermetropia or hypermetropic astigmatism—and are promptly relieved by correction of these defects. If one is in doubt as to the nature of the pain, the instillation of atropine in the eyes is advisable, since rest of the eyes will stop the headache if it is due to ametropia.

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72. Skillern, R. H.: Headache: The Nasal Aspect, Bull. New York Acad. Med. **1**:133, 1925.

In estimating the severity of pain, the statement of the sufferer is not always reliable. Some patients as a matter of pride underestimate their pain, while others, in order to obtain relief by impressing the oculist, magnify their distress. The facial expression and bodily manifestation are of value; the complaint of persistent "agonizing pain" with a cheerful countenance does not square with facts.

The treatment of ocular headache should first be directed toward the relief of asthenopia by glasses. This should be done under a cycloplegic. Bahn,[73] in a series of approximately 100 cases, found it impossible to prescribe proper glasses in one half of them without a cycloplegic.

The heterophoric symptoms should be combated by properly directed prism exercises or prismatic glasses. Van der Brugh [71] claimed to have cured in three years 58 out of 71 persons with headache due to muscle imbalance by ordering prisms (some of these cures might have been effected by prism practice).

Careful refraction brings relief to about 90 per cent of patients with ocular cephalalgia; on the other hand, careless refraction, such as omitting cylinders when astigmatism is present or ordering cylinders in cases in which there is no astigmatism, increases pain. To prove this, Snell [74] suggested the placing of a small cylinder in front of the eye of an emmetropic patient to render him astigmatic; if the patient wears such glasses a few days he will return with headache or other symptoms of ametropia. Ill-fitted frames may cause headache, even after the most careful refraction.

The relief of headache by glasses does not always come as soon as the cause is removed. Prolonged eyestrain sets up a pathologic condition, which requires, like other inflamed parts, time to heal; the accommodative function, acting under the same nerve impulse as that of convergence, has undergone changes which take time for adjustment.

The fact that cranial pain disappears after a person wears glasses for a while, and does not return after the patient stops wearing them, as is occasionally noticed with students during examination, does not prove that glasses were not needed. It shows that rest, improved health and lessened ocular work removed the exciting cause.

In endeavoring to relieve headache, one must always bear in mind that the living eye cannot be treated as an optical instrument, independent of the rest of the body. The eye is a part of an intricate system and is dependent on the physical condition of the body. Attention, therefore, should be paid to the general health of the patient. Inflammations

73. Bahn, C. A.: Headaches of Ocular Origin, New Orleans M. & S. J. **85**:177 (Sept.) 1932.

74. Snell,[67] p. 20.

of the eye and its appendages must be abated and nervous irritation quieted, for it often happens that a diseased lid or conjunctiva or a nervous condition prevents the disappearance of cranial pain after a most careful correction, and in insomnia complete relief of pain in the head is unlikely without adequate restful sleep.

One must be careful, however, not to attribute every case of headache to ocular causes and order glasses as a panacea. Ametropia does not always produce cranial pain, no more than it is always responsible for eyestrain. Ordering glasses for headache in a case of tumor of the brain is as absurd as draining the gallbladder of a person with uncorrected hyperopia; yet, both of these clinical tragedies have been observed.

Finally, it is of interest to note that ocular headache is decreasing both in frequency and in severity, thanks to the better care given to children's eyes at public schools and to the advanced methods of modern ophthalmology. When the patient calls after a lapse of years for a reexamination, he complains more often of disturbed vision or inflammatory symptoms than of headache. Of 572 patients who were reexamined, only 169 presented symptoms of cranial pain and these were of a mild character, while in the original examination headache was the chief or only symptom present and was of a more severe type.

CONCLUSIONS

Ocular headache is situated in the cranial muscles and is due to an abnormal nervous condition in the eye itself.

The severity of headache depends on the degree of functional abnormality of the ocular nerves.

The most trifling defects of the ocular muscles may cause the worst headache in persons whose ocular nerve supply is at fault.

Patients troubled with cephalalgia, as a rule, have good vision; the worst headaches are present in patients with low errors of retraction and fixation.

Cranial pain is rare in persons with functional monocularity and amblyopia, in extremely cross-eyed subjects and in those whose vision is greatly impaired.

Patients troubled with asthenopic headache are, as a rule, free from congestive symptoms of the eyes or lids. Persons suffering from inflammatory symptoms as a result of ocular anomalies rarely have headache; these symptoms, as a rule, do not go together.

About 60 per cent of all persons with ocular conditions and 70 per cent of all persons who require refraction are troubled with cranial pain.

About 60 per cent of all cases of headache are hereditary; children inherit from their parents the anatomic peculiarities responsible for asthenopia.

Headache is most frequent in adult life, between the ages of 20 and 45 years. About 57 per cent of patients complaining of ocular headache are women and 43 per cent are men.

About 80 per cent of all asthenopic headaches are due to ametropia; 20 per cent, to muscular anomalies.

About 15 per cent of all asthenopic cases are marked by inflammatory symptoms of the eye or its appendages or both.

Careful refraction will do away with cephalalgia in 90 per cent of patients, if it is due to eyestrain. Careless refraction will produce or aggravate cranial pain.

The relief of headache by glasses does not always come at once; with some patients it takes time to get rid of the headache habit.

The fact that some patients after a prolonged rest lay aside their glasses with the disappearance of headache is no indication that glasses were not needed; it merely proves that decreased work, improved health and ocular rest enabled the muscles to overcome the strain.

Owing to the better care given to the eyes of school children and the improved methods of modern ophthalmology, the occurrence of headache is on the decline.

The diagnostic value of ocular headache lies in cases in which the eye is not suspected of any abnormalities, in which vision is normal and in which inflammatory symptoms are not present. In such cases headache is the first clue to the existence of asthenopia.

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EFFECT ON INTRA-OCULAR TENSION OF CORNEAL MASSAGE WITH THE TONOMETER OF SCHIÖTZ

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Schiötz himself found that tonometry repeated at short intervals lowered the tension of the normal eye. The indentation of the cornea produced by the plunger of the tonometer reduced the volume of the contents of the globe either directly or by momentarily increasing the intra-ocular tension, resulting in the pressing out of intra-ocular fluid. The after-effect in either case must be a relative hypotony. Procedures of this kind used to play an important rôle in the therapy of glaucoma. During the last twenty years they have become a feature more of differential diagnostic than of therapeutic interest. In their study three possible variables have to be considered: (*a*) the pressure-increasing or volume-reducing stimulus (massage with the finger or the application of weights [tonometer] or forces [dynamometer, pressure bandage]) to the cornea or sclera; (*b*) the subsequent drop in tension, and (*c*) the restitution of normal intra-ocular tension.

It is the object of this paper to consider whether the reaction of the normal human eye to a stimulus of the aforementioned kind is constant and characteristic enough to serve as an aid in the differential diagnosis between the normal and the glaucomatous eye. This is especially important because the work of Müller [1] has shown that the normal eye under physiologic circumstances may, according to the new scale of Schiötz (1924), on a single tonometric determination have a tension of 35 mm. of mercury. A search for further criteria concerning the limits of normal tension was therefore indicated.

The use of the tonometer as a volume-decreasing stimulus has been practiced before. Priestley Smith [2] stated: "The eye is softer after application of a tonometer by expression of fluid from the anterior chamber. Absence of this softening is apparently a nearly constant

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From the first eye clinic of the University of Vienna (Prof. Dr. J. Meller, director) and the eye clinic of the University of Chicago (Dr. E. V. L. Brown, director).

1. Müller, H. K.: Arch. f. Augenh. **104**:89, 1931.
2. Smith, P.: Ophth. Rev. **34**:65 (March) 1915.

indication of obstructive changes in the filtration angle." Polack-van Gelder [3] used a 5.5 or 7.5 Gm. weight and made from seven to fifteen applications four or five seconds apart, usually until a constant reading was obtained. Occasionally she let the tonometer rest on the cornea for a while. Wegner [4] worked with both of these methods, giving the latter, "protracted" tonometry, the preference because of its greater accuracy. In tests on fifty persons with normal eyes, he kept the tonometer on the cornea for from five to six minutes and found that "in many cases the tension dropped one-half, in all of them at least one-third of the original value." The drop in tension was confined to the eye tested, and the age of the patient did not seem to influence it. The depth of the anterior chamber, as measured with the Ulbrich drum, remained the same in most of the cases, though it decreased slightly in a very few (maximum difference in depth, 0.4 mm.). In eyes with light fundi, Wegner thought that he could see a general narrowing of the visible vessels in the choroid. Miotics and mydriatics had no influence on the effect of the corneal massage. Caffeine given intravenously before the beginning of the massage lessened the drop in tension. Wegner explained the effect of corneal massage mainly by the outflow of fluid, most likely blood, from the eye.

The observations of Polack-van Gelder and Wegner on glaucomatous eyes are of interest. The former found no (or very little) reduction of tension by corneal massage in glaucomatous eyes with normal tension, but Wegner could not entirely confirm this observation.

Bailliart,[5] in a lecture on tonometry, also took up the variations in tension during or after the application of the tonometer:

> Most frequently the tension falls after tonometric application. This fall of the tension is certainly comparable to the one described by many authors after prolonged application of my dynamometer. But the reaction I have in view now is different because it is immediate. The needle has scarcely pointed to a figure than it goes down, sometimes by several divisions under that figure. . . . It scarcely takes the needle half a minute to accomplish this movement after which the needle will stop or only go down very slowly. This kind of reaction to tonometric application is by far the most frequent. It is a normal and consequently favourable reaction. It proves that the eye accommodates itself to a new pressure. It comes probably from a modification of the choroidal vascular system that allows itself to be flattened; it is one of its physiological properties to act as a pad regulating intra-ocular pressure.

In some eyes Bailliart found the tension to remain stationary on application of the tonometer.

> This phenomenon occurs with eyes appearing perfectly normal; it occurs too in severe forms of glaucoma. We can, to explain it, suppose that the choroidal

3. Polack-van Gelder, R.: Klin. Monatsbl. f. Augenh. **49**:592, 1911.
4. Wegner, W.: Ztschr. f. Augenh. **55**:381, 1925.
5. Bailliart, P.: Tr. Ophth. Soc. U. Kingdom **51**:412, 1931.

pad has lost its normal elasticity, or that the blood-pressure is abnormally high while in the average eye it is scarcely higher than the venous ocular pressure,[6] or that there is an obstacle to the venous circulation. This deals with cases in which the prognosis is less favourable.

Shope,[7] in his "Study in Tonometric Measurements," concluded that "a fall of tension after application of a tonometer to an eye was the rule both with normal and with glaucomatous eyes, seeming to have little, or no, relation to obstructive changes in the filtration angle."

The discrepancies between the findings of the various authors cited here and the actual lack of quantitative and statistical data on the behavior of the normal eye were the reasons why we undertook this work, which was started in the fall of 1929.

We found phase c very difficult to study in a normal eye because of the repeated tonometric determinations required to obtain a curve giving the process of recovery. We therefore confined ourselves to the determination of phase b.

As to the mechanism of phases b and c, we refer the reader to the excellent paper by Magitot [8] on this subject. The controversy between Sugasawa [9] and Koyanagi [10] as to whether or not the intra-ocular pressure begins to drop while the tonometer still rests on the cornea does not concern us here. We agree with Koyanagi, since Sugasawa has not proved conclusively that an increasing swing of the tonometer lever while the instrument rests on the cornea means no lowering of the intra-ocular tension. We admit, however, that an increase in impressibility of the cornea under the influence of the protracted indentation is not impossible. We are aware of this possibility, namely, that the second tonometric measurement in our experiments, the so-called final tension, may express the actual drop in tension plus the effect of altered impressibility of the cornea.

METHOD

The original stimulus was the indentation of the cornea by the weight of the plunger. To make the stimulus equivalent in all the eyes tested it would have been necessary to vary the weight of the plunger in keeping with the volume of the globe, with the impressibility of the cornea, which is known to vary greatly with age (Müller [11]), and with the

6. This clause is apparently misprinted and should read: ". . . or that the venous blood-pressure is abnormally high while in the average eye it is scarcely higher than the ocular pressure. . . ."

7. Shope, P.: Am. J. Ophth. **15**:739, 1932.

8. Magitot, A.: Ann. d'ocul. **168**:785, 1931.

9. Sugasawa, N.: Klin. Monatsbl. f. Augenh. **77**:775, 1926.

10. Koyanagi, Y.: Klin. Monatsbl. f. Augenh. **78**:616, 1927.

11. Müller, H. K.: Arch. f. Augenh. **105**:504, 1932.

intra-ocular tension, which is likely to vary with age and, indeed, with other variables (Jackson [12]). Unfortunately we have no reliable data concerning some of these factors, and as it was our plan to make the test simple enough to be used in clinical ophthalmology, we gave up the idea of an equivalent stimulus and chose a stimulus which in absolute measures remained the same in all experiments. Following a suggestion by Seidel, we chose the Schiötz tonometer with the 15 Gm. weight and let it rest on the patient's cornea for exactly two minutes. The stimulus could therefore be called 30 gram-minutes. The intra-ocular tension was determined immediately before and immediately after the massage.

We found that it was fairly easy for the patient to hold his eyes still for two minutes. The deflection of the lever varied from 9.5 to 13 scale units, corresponding to indentations of from 0.48 to 0.65 mm. in depth. The minimum drop in tension was 5 mm., which is well beyond the limit of error for two tonometric determinations on the same eye only a few minutes apart. In a few experiments the tonometer was kept on the cornea for three and four minutes and the tension recorded every minute. Most of the drop occurred during the first two minutes, and only a little of it during the third and fourth minutes. Some of the patients became nervous during the third minute. This fact and our desire to lessen the insult to the cornea led us to choose a period of two minutes as most suitable for diagnostic corneal massage.

The patients were chosen from those in the outpatient department and from the hospitalized patients of the first eye clinic of the University of Vienna. For all of them it was the first tonometric determination. Only patients with errors of refraction and without any active inflammatory process or gross sign of glaucoma were used. It is possible that some of the eyes tested would on provocative tests or extended observation have shown signs of glaucoma. One eye after the other was subjected to the procedure described.

The corneal massage usually produced a slight temporary depression (*Eindellung*) of the epithelium in the area which had been in contact with the plunger. This area, however, did not stain with fluorescein. The patients were dismissed without dressings shortly after the test. No complication was observed.

RESULTS

In every one of the "normal" eyes tested the tension dropped under the influence of protracted tonometry. The decrease varied from 5 to 15 mm. of mercury (table 1).

The original tension varied from 7 to 32 mm. of mercury. In table 2 we have classified the original tensions of the normal eyes in groups of

12. Jackson, E.: Ophthalmology **13**:437 (April) 1917.

Case	Age	Sex	Unusual Features		Corrected Vision*		Right Eye			Left Eye					
			Right Eye	Left Eye	Right Eye	Left Eye	*a* Original Tension, Mm.	*b* Final Tension, Mm.	*c* Drop, Mm.	*d* Original Tension, Mm.	*e* Final Tension, Mm.	*f* Drop, Mm.	*a* − *d*	*b* − *e*	*c* − *f*
1	42	F	Incipient cataract	.....	6/12; Jg. 2	6/12; Jg. 2	24	17	7	22	17	5	+ 2	0	+ 2
2	52	F	Anterior chamber shallow	Anterior chamber shallow	6/6; Jg. 1	6/6; Jg. 1	31	19	12	31	14	17	0	+ 5	− 5
3	24	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	15	7	22	15	7	0	0	0
4	48	F	Inferior conus	Inferior conus	6/6; Jg. 1	6/6; Jg. 1	28	17	11	30	18	12	− 2	− 1	− 1
5	67	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	28	18	10	30	19	11	− 2	− 1	− 1
6	38	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	23	15	8	23	15	8	0	0	0
7	42	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	28	20	8	28	20	8	0	0	0
8	65	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	27	17	10	27	17	10	0	0	0
9	55	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	31	15	16	31	15	16	0	0	0
10	18	F	.....	.....	6/6(?); Jg. 1	6/6(?); Jg. 1	28	16	12	28	16	12	0	0	0
11	50	F	.....	.....	6/8; Jg. 1	6/8; Jg. 1	26	17	9	30	20	10	− 4	− 3	− 1
12	46	F	Anterior chamber very shallow	Anterior chamber very shallow	6/6; Jg. 1	6/6; Jg. 1	27	19	8	31	21	10	− 4	− 2	− 2
13	52	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	28	19	9	22	16	10	+ 6	+ 3	+ 3
14	25	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	26	13	13	26	13	13	0	0	0
15	24	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	28	16	12	26	15	11	+ 2	+ 1	+ 1
16	50	F	.....	Inferior conus	6/6; Jg. 1	6/18; Jg. 2	26	16	10	30	18	12	− 4	− 2	− 2
17	23	M	.....	Paresis of fourth nerve	6/6-6/5; Jg. 1	6/6-6/5; Jg. 1	22	11	11	22	11	11	0	0	0
18	50	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	14	8	22	14	8	0	0	0
19	35	F	.....	Paresis of fifth, sixth and seventh nerves	6/6; Jg. 1	6/6; Jg. 1	26	15	11	26	15	11	0	0	0
20	59	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	14	8	22	14	8	0	0	0
21	24	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	11	11	20	11	9	+ 2	0	+ 2
22	15	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	14	8	22	13	9	0	+ 1	− 1
23	42	M	.....	.....	6/8; Jg. 1	6/8; Jg. 1	22	13	9	19	11	8	+ 3	+ 2	+ 1
24	31	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	14	8	24	14	10	− 2	0	− 2
25	59	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	14	8	26	15	11	− 4	− 1	− 3
26	27	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	29	17	12	31	17	14	− 2	0	− 2
27	21	M	.....	.....	6/8; Jg. 1	6/6; Jg. 1	29	17	12	25	15	10	+ 4	+ 2	+ 2
28	22	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	13	9	22	13	9	0	0	0
29	62	M	Inferior conus	Nasal conus	6/6; Jg. 1	6/6; Jg. 1	26	16	10	26	16	10	0	0	0
30	38	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	14	8	22	14	8	0	0	0
31	54	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	26	15	11	26	15	11	0	0	0
32	27	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	30	15	15	22	11	11	+ 8	+ 4	+ 4
33	46	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	27	19	8	31	21	10	− 4	− 2	− 2
34	14	M	.....	.....	6/8; Jg. 1	6/8; Jg. 1	26	18	8	30	18	12	− 4	0	− 4
35	52	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	28	19	9	22	16	6	+ 6	+ 3	+ 3
36	23	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	26	17	9	26	17	9	0	0	0
37	62	M	Nuclear cataract	.....	6/24; Jg. 1	6/6; Jg. 1	26	19	7	22	17	5	+ 4	+ 2	+ 2
38	65	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	31	16	15	27	15	12	+ 4	+ 1	+ 3
39	46	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	29	16	13	31	19	12	− 2	− 3	+ 1
40	44	F	Maculae corneae	Maculae corneae	6/8; Jg. 1	6/8-6/6(?); Jg. 1	31	15	16	31	15	16	0	0	0
41	29	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	12	10	20	11	9	+ 2	+ 1	+ 1
42	32	F	.....	.....	6/8; Jg. 1	6/8; Jg. 1	19	11	8	19	11	8	0	0	0
43	24	M	.....	.....	6/12; Jg. 1	6/6; Jg. 1	26	14	12	26	14	12	0	0	0
44	28	M	Temporal conus	Temporal conus	6/8; Jg. 1	6/8; Jg. 1	28	15	13	28	15	13	0	0	0
45	21	M	.....	.....	6/12; Jg. 1	6/6; Jg. 1	30	15	15	28	15	13	+ 2	0	+ 2
46	55	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	12	10	19	11	8	+ 3	+ 1	+ 2
47	45	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	11	11	26	12	14	− 4	− 1	− 3
48	22	M	.....	.....	6/6; Jg. 1	6/6; Jg. 1	26	15	11	24	14	10	+ 2	+ 1	+ 1
49	41	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	22	13	9	24	13	11	− 2	0	− 2
50	17	F	.....	.....	6/6; Jg. 1	6/6; Jg. 1	20	15	5	20	15	5	0	0	0

* Jg. 1 and Jg. 2 indicate ability to read Jaeger's test types no. 1 and no. 2, respectively.

3 mm. Within those groups we have listed the respective decreases in groups of 1 mm.

The following questions arose:

1. Does the decrease in tension depend on the original tension of the eye tested?

2. Does it depend on the depth of the impression produced with the tonometer?

3. Does it depend on the age of the patient?

4. Is it constant for the same person or for the same eye?

To decide the first question, it was necessary to determine the correlation between the original tension and the amount of decrease. A positive correlation coefficient of 0.74 was obtained, which indicates a strong tendency of high original tensions to be coupled with the greater

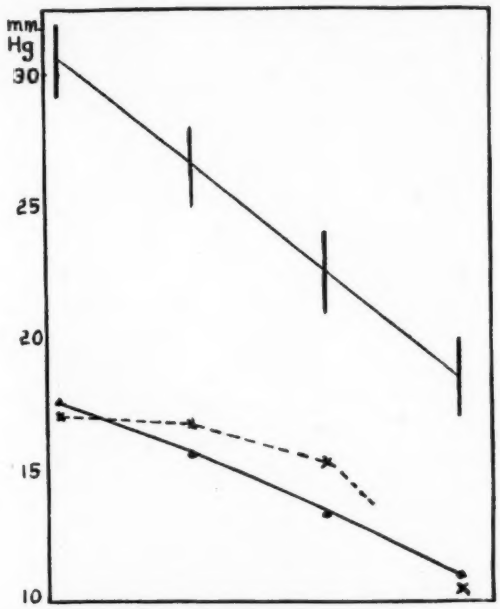
TABLE 2.—*Original Tensions of Normal Eyes* *

	Original Tension, Mm.				
Decrease in Tension, Mm.	32-29	28-25	24-21	20-17	
17	1 (1)	.....	.....	.....	
16	4 (2)	.....	.....	.....	
15	3 (1)	.....	.....	.....	
14	1	1	.....	.....	
13	1	5	.....	.....	
12	7 (2)	6 (1)	.....	.....	
11	1 (1)	8 (3)	6	.....	
10	3 (1)	7 (6)	4 (1)	.....	
9	.....	5 (3)	5	2	
8	.....	5	11 (5)	4 (1)	
7	.....	1 (1)	3	.....	
6	.....	.....	2 (2)	.....	
5	.....	.....	2 (1)	2	
Total	21 (8)	38 (14)	33 (9)	8 (1)	100 (32)

* The figures in parentheses indicate the number of eyes of persons over 50.

decreases of tension. If the eyes are put in groups according to the original tension (table 2), this tendency is clearly brought out. The greater decreases occur more frequently in eyes in which the tension is high before the massage. The greatest decreases occur only in eyes with high, and the smallest decreases only in eyes with low, original tension. This behavior is apparently characteristic for the normal eye. If one accepts Wegner's and Bailliart's opinion that the decrease results from outflow of blood from the eye, the greater decrease in the eye with high original tension suggests that this type of eye contains more blood than can readily be displaced, perhaps even that the cause of the high normal tension in the normal eye is that there is a large amount of blood which can easily be displaced. Squeezing of the eye can hardly be a source of error in these experiments. A great drop in tension in eyes with high original tension occurred too consistently in both eyes of the same person to be explained on the basis of squeezing which is capable of producing an unduly high tension before and after corneal massage. The answer to question 1 is therefore that the decrease in tension does depend on the original tension of the eye tested.

The definite positive correlation between the original tension and the decrease also answers question 2. The impression produced by the tonometer was the greatest in the eyes with low original tension. The deformation of the cornea was the deepest and greatest in the eyes in which the drop in tension was the smallest. The second question has thus to be answered in the negative. The depth of the impression in our experiments on normal eyes was not of great significance. This was another reason why we felt that a constant weight of the plunger, i. e., a varying depth of indentation, was permissible.



The short heavy vertical lines indicate the limits of the groups established in table 2. The lower continuous line gives the arithmetical means of the final tension in each of those groups for persons below 50, and the broken line, the corresponding figures for persons over 50. The lower continuous line is almost straight, suggesting a definite constant ratio between the original and the final tension.

Question 3 was more difficult to answer. It is well known that the same indentation produced with the tonometer in the eyes of older people means a different, namely, a lower, intra-ocular tension. From the data available at this time (Müller [11]) it is impossible to draw a sharp line between the "normal" and the "senile" eye in this respect. Therefore, we treated the eyes of persons over 50 in the same way as those of younger patients and expressed the findings according to the general scale of Schiötz. We realize that the deflections of the tonometer in tests on patients over 50 most likely meant a lower absolute tension than was indicated by the scale of Schiötz. This, however, would

merely have put those eyes in a group with a lower original tension and a smaller absolute decrease without changing the ratio of the original tension to the decrease.

In table 2 the number of eyes of persons over 50 is given in brackets after the figure for the total number of eyes in each group. There

TABLE 3.—*Results of Repeated Massage Tests on Three Patients*

Name	Age	Sex	Time of Observations	Original Tension, Mm.		Diameter of Pupil, Mm.		Tension After Massage Test, Mm.	
				R. E.	L. E.	R. E.	L. E.	R. E.	L. E.
H. H.	31	F	8/10/30 9:00 a.m.	27	27	4.0	4.0	..	..
			4:00 p.m.	21	20	5.5	5.0	..	..
			8:00 p.m.	20	20	4.5	4.5	..	..
			8/11/30 8:00 a.m.	20	20	5.5	6.0	..	..
			4:00 p.m.	19	18	3.5	4.0	..	..
			8/12/30 8:00 a.m.	19	20	4.0	4.0	..	..
			8/13/30 3:00 p.m.	21	20	4.5	4.0	..	..
			8/14/30 7:00 a.m.	18	18	...	...	..	11
			8:00 p.m.	18	19	4.5	4.5	..	11
			8/15/30 7:30 a.m.	16	18	3.5	4.5	..	11
			2:00 p.m.	..	..	3.5	4.0	..	..
			8/16/30 8:30 a.m.	17	15	5.0	4.5	..	10
			7:00 p.m.	16	17	...	...	..	13
			8/17/30 7:30 a.m.	18	19	5.5	6.0	..	..
			4:00 p.m.	17	17	4.0	4.0	..	..
			8/18/30 8:00 a.m.	18	19	4.0	4.0	..	..
			4:00 p.m.	16	18	4.5	4.5	..	..
			8:00 p.m.	16	15	5.0	5.0	..	..
F. K.	45	F	9/ 8/30 8:00 a.m.	22	22	3.5	3.75	..	..
			2:00 p.m.	20	20	4.0	4.0	..	..
			8:00 p.m.	18	19	3.5	3.5	..	..
			9/ 9/30 8:00 a.m.	25	23	3.0	3.0	..	..
			9/10/30 8:00 a.m.	20	20	2.5	2.5	..	..
			4:00 p.m.	20	20	2.5	2.5	..	12
			9/11/30 8:00 a.m.	19	18	2.5	2.5	..	13
			4:00 p.m.	23	23	3.5	3.5	..	..
			8:00 p.m.	20	18	4.0	3.5	..	12
			9/12/30 10:00 a.m.	19	19	3.0	3.0	..	10
			4:00 p.m.	18	19	2.5	2.5	..	..
A. F.	44	F	11/7/30 8:30 a.m.	22	18	2.5	2.5	..	..
			12:30 p.m.	20	19	3.0	2.75	..	..
			4:00 p.m.	18	14	3.75	3.50	..	..
			9:00 p.m.	16	15	2.75	2.50	..	..
			11/8/30 8:00 a.m.	16	16	3.0	2.75	..	11
			4:00 p.m.	18	18	3.0	3.0	..	..
			7:00 p.m.	18	18	3.25	3.0	..	9
			11/9/30 9:30 a.m.	19	16	3.0	2.75	..	9
			7:00 p.m.	16	12	3.0	2.75	..	9

seems to be no tendency of the eyes of older persons to show small drops in tension more frequently than those of persons under 50. The accompanying chart shows no considerable difference in the mean drop of tension between persons under and those over 50.

Finally we studied question 4, as to how constant the result of the massage test is for the same eye at different times or for the two eyes of the same person. In table 1 we compared the original tensions, final tensions and drops of tension in the eyes of the same patient. Wegner

expressed the belief that corneal massage of one eye does not alter the tension of its mate. We therefore felt that it was safe to massage one eye shortly after the other.

Only three of our fifty patients (6 per cent) showed a difference of more than 3 mm. of mercury in the decrease in tension in the two eyes. In two of these patients the original tensions were markedly different, so that a different result of corneal massage had to be expected. In 94 per cent of the patients the reductions in tension produced by corneal massage in the two eyes either were the same or showed a difference of less than 3 mm.

In three patients seen in the Albert Merritt Billings Hospital of the University of Chicago we had the opportunity of repeating the massage test several times (table 3).

The reaction of the normal human eye, therefore, seems—within the limits shown in this paper—to be constant and characteristic.

EFFECT OF PROLONGED TONOMETRY ON GLAUCOMATOUS EYES

In order to get some idea about the behavior of the glaucomatous eye, we subjected to the massage test a few patients with definite primary glaucoma who had been under our observation for longer periods and had been rather thoroughly studied. Only eyes in which the original tension was 36 mm. or less at the time of the massage tests were considered in this connection. If the tension was over 36 mm., we felt that the volume-decreasing stimulus was probably too small and the reaction of the eye to it not quite comparable with the reactions of eyes with lower tensions.

We found that in the glaucomatous eyes with original tensions of 36 mm. or less the result of the massage test depended mainly on the original tension. If the original tension was below 27 mm. most of the eyes with simple or chronic inflammatory glaucoma, in early or late stages and apparently independent of medication, showed decreases of the order found in normal eyes with the same original tensions. The decrease was occasionally 1 or 2 mm. below the lower limit for the respective group, but not consistently so. In those cases, therefore, the massage test in the form in which we used it allowed no differentiation between normal and glaucomatous eyes. There may have been a slight quantitative difference, but if so it was too small to be recognized with certainty.

In the group of eyes with original tensions between 27 and 36 mm. we found that most of the eyes showed drops considerably lower than the lower limit for normal eyes of approximately the same tension. In this group, therefore, the massage test seemed to suggest a difference between the normal and the glaucomatous eye. Since in this group of

eyes with "high normal" tensions the differential diagnosis between glaucoma and no glaucoma is of the greatest importance, we felt that the massage test might be of some use. How valuable it actually is can be determined only by applying it to a large number of eyes, which we hope to be able to do in the near future.

The behavior of glaucomatous eyes with high normal tensions on which fistulizing operations had been performed was different from that of glaucomatous eyes that had not been operated on as the drops in tension frequently were of the same magnitude as in the normal eye.

In one eye with secondary glaucoma following anterior uveitis we made the observation that an original tension under 27 mm. consistently dropped less than the corresponding tension in a normal eye. If the original tension was over 27 mm., the eyes with secondary glaucoma behaved like the ones with primary glaucoma. Unfortunately we were not able to get any data on eyes with obstruction of the central vein before the secondary glaucoma set in. Among eyes that "appeared normal" (Bailliart) we did not find any in which the tension did not drop to the extent outlined in this paper for the normal eye. We could therefore not confirm the observation of Bailliart ("loss of elasticity of the choroidal pad in an otherwise normal eye"—a condition which may be very rare).

We feel that the corneal massage test in the form described may be of some use in the diagnosis of glaucoma and perhaps of vascular diseases. Our observations on a larger number of eyes with pathologic conditions will be published at a later date.

SUMMARY

The behavior of the intra-ocular tension of normal eyes in response to the application for two minutes of a tonometer carrying a 15 Gm. weight was studied on fifty persons. In every case the tension dropped in proportion to the original tension. Normal eyes with tensions between 27 and 32 mm. showed a greater decrease than a small number of glaucomatous eyes with the same original tensions.

CALCULATING STEREOSCOPIC VERGENCE

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The stereoscope in one form or another is still extensively used in modern orthoptic training. Often instead of the usual stereoscope a stereoscope-like arrangement can be obtained by utilizing the ordinary instruments required for refraction. The essential parts of the refracting stereoscope are a pair of prisms with the bases out, a septum, a card-holder and preferably a pair of convex lenses. This arrangement, obtained by combining parts of one's refracting equipment, has greater adaptability than the regular stereoscopes. The strength of the prism power, as well as the strength of the lenses used, can be varied at will. In the usual Holmes stereoscope the prisms are O. U. 8.00 Δ and the lenses O. U. + 5.25.

Whatever stereoscope or stereoscopic arrangement is used, it is desirable for an intelligent use of the instrument in orthoptic training that the physician know at all times the state of the patient's vergence, i. e., the amount of convergence or divergence when corresponding points are fused on the stereogram. Otherwise he is apt to work more or less blindly and lose some fine points in stereoscopic technic. This is especially so when the stereoscopic arrangement is such that the halves of the stereogram can be brought closer together or separated any desired distance and the vergence as a result constantly changes.

There are several ways of calculating the state of the patient's vergence in any given condition. The method presented here has been found, on the whole, the simplest and most satisfactory, and, in my experience, the one most easily learned.

I shall call the direction of the two visual axes when corresponding points are fused on a stereogram the stereoscopic vergence or, briefly, the stereovergence. If the visual axes are parallel the stereovergence is zero. If the visual axes are convergent (with reference to a state of parallelism) the stereovergence is plus; that is, there is stereoconvergence. If the visual axes are divergent (with reference to a state of parallelism) the stereovergence is minus; that is, there is stereodivergence.

The amount and nature of the stereovergence at any time, whether plus or minus, depends primarily on the following factors: (*a*) the strength of prisms used in the stereoscope, (*b*) the interpupillary distance, (*c*) the distance between the corresponding points on the stereogram and (*d*) the distance of the stereogram from the eyes. Of these, (*b*) is practically a fixed quantity; (*a*), (*c*) and (*d*) are variables.

THE COMPONENTS OF STEREOVERGENCE

The stereovergence is made up of two elements: (1) the prism vergence and (2) the chart vergence. The prism vergence is the vergence produced by the prisms used and is always plus, as the prisms are placed with the bases out and have a convergent effect. In the simple hand stereoscope the prism vergence is plus 16 Δ, i. e., 16 Δ convergence. The chart vergence is the vergence required in order to fuse the two corresponding points without any prisms. Thus, if the separation of the corresponding points on the chart is 6 cm. and P. D. (the interpupillary distance) is 6 cm., the chart vergence is zero, as the parallel visual axes fixate the corresponding points. If the separation of the corresponding points on the chart is 5 cm. and P. D. is 6 cm., some convergence of the eyes is necessary in order to fixate the corresponding points. If the separation of the corresponding points on the chart is more than P. D., that is, if the separation is 7 cm. and P. D. is 6 cm., some divergence of the eyes is necessary in order to fixate the corresponding points. Thus the chart vergence may be zero, plus (chart convergence) or minus (chart divergence).

The chart vergence depends on three elements: (*a*) P. D. (the interpupillary distance); (*b*) O. S. (the object separation), that is, the distance between corresponding points on the stereogram, and (*c*) L (the location of the chart), that is, its distance from the eyes. When P. D. is equal to O. S. the chart vergence is zero. Positive or negative chart vergence occurs only when there is a difference between P. D. and O. S. This difference, taken in centimeters, when multiplied by 100 and divided by L (the location or distance of the chart) gives the chart vergence. For example, if P. D. is 6 cm., O. S. 5 cm. (the corresponding points are 5 cm. apart) and L 20 cm. (the stereogram is at a distance of 20 cm.), the chart vergence is 100 times the difference between P. D. and O. S., which is 100 times L divided by 20. This gives 5 Δ of chart convergence.

In formula this rule reduces to $\Delta = \frac{100d}{L}$, in which Δ equals the number of prism diopters, d the difference between the interpupillary distance (P. D.) and the object separation (O. S.), taken in centimeters, and L the location of the chart, also in centimeters. P. D. can generally be taken as 6 cm. in adults and 5 cm. in small children. If the prisms remain unchanged the stereoscopic vergence can be readily calculated, as the following illustrations show.

Suppose that one is examining an adult. P. D. is 6 cm., using the ordinary stereoscope, with fixed prisms of O. U. 8 Δ, bases out, and the chart is at a distance of 20 cm. The corresponding points are 3 cm. apart, or there are two half charts approximately 3 cm. apart. Then

P. D. is 6, O. S. is 3 and L is 20. The chart vergence is 6 less 3, times 100, divided by 20, or, substituting in $\Delta = \frac{100d}{L}$, $\Delta = 15$; the prism vergence is plus 16 Δ; therefore the total stereovergence is plus 31 Δ, i. e., a convergence of 31 Δ. If the chart is moved to 16 cm. and the separation is increased to 4 cm., what is the stereovergence? O. S. is 4 and L is 16, $\Delta = \frac{100 \times 2}{16}$ = plus 12.5. Add 16 Δ (the prism convergence), and the stereovergence is plus 28.5 Δ. If at the same distance the parts are separated by 5 cm., what is the vergence? O. S. is 5, L is 16 and $\Delta = \frac{100 \times 1}{16}$ = plus 6.25 Δ. Adding plus 16 Δ, the stereovergence is plus 22.25 Δ. If at the same distance the parts are separated by 7 cm., what is the vergence? O. S. is 7 cm., and the difference between the interpupillary distance (P. D.) and the object separation (O. S.) is minus 1 (6—7) : d is minus 1. Therefore $\Delta = \frac{100 \times (-1)}{16}$ = —6.25Δ. Adding plus 16 Δ (the prism convergence), the stereovergence is plus 9.25 Δ. Or, finally, with the chart at 10 cm. and with the parts separated by 8 cm., what is the vergence? d is minus 2 and L is 10; therefore, $\Delta = \frac{100 \times (-2)}{10}$ =minus 20 Δ. Adding plus 16 Δ (the prism convergence), the stereovergence is minus 4 Δ; the eyes diverge 4 prism diopters.

This method of calculating the stereovergence with any stereoscope, with the charts at any distance and with every possible degree of separation between the corresponding points becomes almost second nature after a little practice. Fractions of a prism diopter can be ignored, and in many cases the calculations can be done mentally. This is especially so if the exercises are given with the charts placed at a fixed distance and if the changes in vergence are produced by the separation or approximation of the corresponding points. Thus, in the formula $\Delta = \frac{100d}{L}$, if L is fixed $\frac{100}{L}$ becomes a fixed number or coefficient, which may be called a vergence coefficient, and only d varies. For instance, if the charts are kept at 25 cm. the vergence coefficient is 4, and d is always multiplied by 4. If O. S. is 4 cm., d is 2, and Δ is +8 Δ. If O. S. is 5 cm., d is 1, and Δ is +4 Δ. If O. S. is 6 cm., d is 0, and Δ is 0. If O. S. is 7 cm., d is minus 1, and Δ is minus 4, and if O. S. is 9 cm., d is minus 3, and Δ is minus 12 Δ, and so on.

One can easily find the vergence coefficient for any distance by dividing 100 by that distance in centimeters. Thus the vergence coefficient for 30 cm. is 3. (Fractions may be neglected in mental calculations as they are of little consequence.) It is sufficient to note the vergence coefficients for various distances, as given in table 1.

As a final illustration, if with the B chart of the Wells selection (original Javal M) a child whose P. D. is about 50 mm. (5 cm.) sees the arrow over black ball no. 9, the chart being at 14 cm. and the stereoscopic arrangement having an O. U. of 5 Δ, bases out, what is the stereoscopic vergence? In this instance d is minus 4; (5 cm.—9 cm.), the vergence coefficient is 7, and the chart vergence is minus 28 Δ. The prism vergence is plus 10 Δ. The stereovergence is minus 18 Δ, i. e., a divergence corresponding to 18 Δ, bases in, or about 3.5 meter angles.

The formula $\Delta = \frac{100d}{L}$ is easily obtained from similar triangles and is worth noting because it may be used to check the accuracy in any case. In figure 1, let a equal the interpupillary distance (P. D.) in centimeters, b the distance between corresponding points on the chart (O. S.) and L the location or distance of the chart from the eyes in centi-

TABLE 1.—*Vergence Coefficients for Various Distances of the Chart*

Distance, Cm.	Vergence Coefficient
28–35	3
23–27	4
19–22	5
16–18	6
14–15	7
12–13	8
11	9
10	10

meters. By connecting the extremities of a with those of b, the direction of the visual axes (AB and A′B′) or the extent of their convergence is obtained. These are projected until they meet at M, at a distance x from b. Two similar triangles are formed, and the proportion is $\frac{a}{b} = \frac{L + x}{x}$, which resolves to $x = \frac{bL}{a—b}$. The point at which the visual axes meet is at a distance of L + x, which reduces to $\frac{aL}{a—b}$. The convergence in meter angles is found by dividing 100 by this distance, i. e., 100 divided by $\frac{aL}{a—b}$ gives the number of meter angles. To convert the result into prism diopters (Δ), the number of meter angles is multiplied by P. D. in centimeters, i. e., by a. This works out to $\Delta = \frac{100a\ (a—b)}{aL}$, which reduces to $\Delta = \frac{100\ (a—b)}{L}$. If a—b, the difference between the interpupillary distance and the object separation, is called d, the formula reduces to $\Delta = \frac{100d}{L}$ [formula 1].

To derive the general formula it was necessary to use literal equations which may be unfamiliar to some, but to use the similar method in specific instances is simple. If P. D. (a) is 6 cm., O. S. (b) 5 cm. and L, 15 cm., the proportion is 6:5 = 15 + X:X, and X = 75

cm. The distance from a to M equals 90 cm.; therefore the number of meter angles is $\frac{100}{90}$, and the number of prism diopters is this fraction multiplied by 6, which gives $\frac{100 \times 6}{90}$, or $6\frac{2}{3}$ Δ.

The formula $\Delta = \frac{100d}{L}$ was derived in a case in which the P. D. was greater than O. S., necessitating convergence. To show that this formula is general, a case may be considered in which P. D. is less than O. S., necessitating divergence.

In figure 2, a again equals P. D. in centimeters, b the distance between corresponding points on the chart and L the location or distance

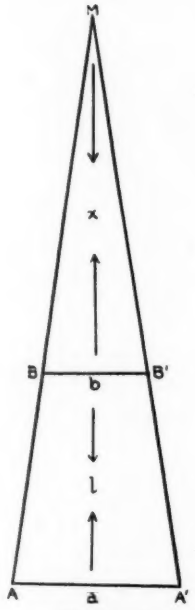

Figure 1

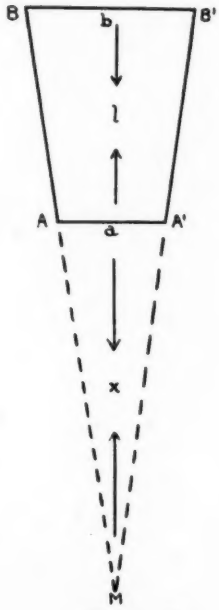

Figure 2

Fig. 1.—Geometric illustration of the principle of stereoscopic vergence: *a* indicates the interpupillary distance; *b*, the distance between corresponding points on the chart; *l*, the distance of the chart from the eyes, and *M*, the point of fusion of the visual axes *AB* and *A'B'* at a distance *x* from the corresponding points on the chart; *a* is greater than *b*, necessitating convergence.

Fig. 2.—In this instance *a* is less than *b*, necessitating divergence.

of the chart. The visual axes are represented by AB and A'B'. It is evident that the axes must diverge in order to fixate B and B'. By projecting AB and A'B' backward until they meet at M, two similar triangles are formed. The proportion is $\frac{b}{a} = \frac{L+x}{x}$. Solving this equation and continuing in the same manner as for figure 1, the result is: $\Delta = \frac{100\ (b-a)}{L}$ [formula 2].

It will be seen that formula 1 differs from formula 2 by the sign. In 1 the symbol (a—b) occurred; in 2, (b—a). Formula 1 can be used in all cases, however, if the minus sign is used to represent divergence. With this convention, formula 1, written $\Delta = \frac{100d}{L}$, is applicable to all cases. When b is greater than a, d is a minus quantity and represents divergence, as is evident from figure 2.

Those who visualize plus or minus vergence more readily when it is expressed in meter angles than when it is given in prism diopters

TABLE 2.—*Chart Vergences for Calculating Stereoscopic Vergence* *

P. D.†	Distance Between Corresponding Points on Stereogram (Cm.)																			
50.....	...	...	...	2.0	2.5	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0
55.....	...	...	2.0	2.5	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0	
60.....	...	2.0	2.5	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0		
65.....	2.0	2.5	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0			

Ch. D.§	Chart Vergence in Prism Diopters for Various Distances from the Eyes (Cm.)‡																			
	+	+	+	+	+	+	+	+	+		—	—	—	—	—	—	—	—	—	—
10.....	45.0	40.0	35.0	30.0	25.0	20.0	15.0	10.0	5.0	0	5.0	10.0	15.0	20.0	25.0	30.0	35.0	40.0	45.0	50.0
11.....	40.9	36.4	31.8	27.3	22.7	18.2	13.6	9.1	4.5	0	4.5	9.1	13.6	18.2	22.7	27.3	31.8	36.4	40.9	45.5
12.....	37.5	33.3	29.2	25.0	20.8	16.7	12.5	8.3	4.2	0	4.2	8.3	12.5	16.7	20.8	25.0	29.2	33.3	37.5	41.7
13.....	34.6	30.8	26.9	23.1	19.2	15.4	11.5	7.7	3.8	0	3.8	7.7	11.5	15.4	19.2	23.1	26.9	30.8	34.6	37.7
14.....	32.1	28.6	25.0	21.4	17.9	14.3	10.7	7.1	3.6	0	3.6	7.1	10.7	14.3	17.9	21.4	25.0	28.6	32.1	35.7
15.....	30.0	26.7	23.3	20.0	16.7	13.3	10.0	6.7	3.3	0	3.3	6.7	10.0	13.3	16.7	20.0	23.3	26.7	30.0	33.3
16.....	28.1	25.0	21.9	18.8	15.6	12.5	9.4	6.3	3.1	0	3.1	6.3	9.4	12.5	15.6	18.8	21.9	25.0	28.1	31.3
17.....	26.5	23.5	20.6	17.6	14.7	11.8	8.8	5.9	2.9	0	2.9	5.9	8.8	11.8	14.7	17.6	20.6	23.5	26.5	29.4
18.....	25.0	22.2	19.4	16.7	13.9	11.1	8.3	5.6	2.8	0	2.8	5.6	8.3	11.1	13.9	16.7	19.4	22.2	25.0	27.8
19.....	23.7	21.1	18.4	15.8	13.2	10.5	7.9	5.3	2.6	0	2.6	5.3	7.9	10.5	13.2	15.8	18.4	21.1	23.7	26.3
20.....	22.5	20.0	17.5	15.0	12.5	10.0	7.5	5.0	2.5	0	2.5	5.0	7.5	10.0	12.5	15.0	17.5	20.0	22.5	25.0
21.....	21.4	19.1	16.7	14.3	11.9	9.5	7.1	4.8	2.4	0	2.4	4.8	7.1	9.5	11.9	14.3	16.7	19.1	21.4	23.8
22.....	20.5	18.2	15.9	13.6	11.4	9.1	6.8	4.5	2.3	0	2.3	4.5	6.8	9.1	11.4	13.6	15.9	18.2	20.5	22.7
23.....	19.6	17.4	15.2	13.0	10.9	8.7	6.5	4.3	2.2	0	2.2	4.3	6.5	8.7	10.9	13.0	15.2	17.4	19.6	21.7
24.....	18.8	16.7	14.6	12.5	10.4	8.3	6.3	4.2	2.1	0	2.1	4.2	6.3	8.3	10.4	12.5	14.6	16.7	18.8	20.8
25.....	18.0	16.0	14.0	12.0	10.0	8.0	6.0	4.0	2.0	0	2.0	4.0	6.0	8.0	10.0	12.0	14.0	16.0	18.0	20.0
26.....	17.3	15.4	13.5	11.5	9.6	7.7	5.8	3.8	1.9	0	1.9	3.8	5.8	7.7	9.6	11.5	13.5	15.4	17.3	19.2
27.....	16.7	14.8	13.0	11.1	9.3	7.4	5.6	3.7	1.9	0	1.9	3.7	5.6	7.4	9.3	11.1	13.0	14.8	16.7	18.5
28.....	16.1	14.3	12.5	10.7	9.0	7.1	5.4	3.6	1.8	0	1.8	3.6	5.4	7.1	9.0	10.7	12.5	14.3	16.1	17.9
29.....	15.6	13.8	12.1	10.3	8.6	6.9	5.2	3.4	1.7	0	1.7	3.4	5.2	6.9	8.6	10.3	12.1	13.8	15.6	17.2
30.....	15.0	13.3	11.7	10.0	8.3	6.7	5.0	3.3	1.7	0	1.7	3.3	5.0	6.7	8.3	10.0	11.7	13.3	15.0	16.7

* It must be emphasized that the convergence or divergence shown in the table represents chart vergence only. To get the stereoscopic vergence one must add the strength of the stereoscopic prisms used. When the ordinary hand stereoscope is used one must add plus 16 △ because of the O. U. 8△, bases out, embodied in the instrument. Strictly speaking, the prism power in the ordinary stereoscope is not fixed, but depends on the interpupillary distance. The prism effect is obtained from the decentration of the convex lenses used, which are O. U. plus 5.25 or O. U. plus 5.00. With the latter, a prism effect of O. U. 8△ is obtained for the average adult P. D. of 63 mm. For a larger P. D. the prism effect is less; for a smaller P. D. it is more. The change is 1△ from the total of 16△ for every 2 mm.

† Interpupillary distance in millimeters.

‡ The plus signs at the head of the columns indicate plus chart vergence, that is, convergence; the minus signs indicate minus charge vergence, i. e., divergence.

§ Distance of stereoscopic charts from the eyes in centimeters.

can readily convert the stereovergence obtained in prism diopters to meter angles. All that is necessary is to divide the vergence in prism diopters by P. D. in centimeters, which is practically 6 cm. for an adult and 5 cm. for a child.

Table 2 gives the chart vergence for four interpupillary distances, 50, 55, 60 and 65 mm., respectively, for all distances of the charts between 10 and 30 cm. in 1 cm. intervals and for a varying separation of the charts of from 2 to 10 cm. This table can be used in all cases

to obtain the stereoscopic vergence. All that is necessary is to add the prism vergence used in the particular set-up. Thus, if the stereoscopic arrangement carries 10 Δ, bases out (O. U. 5 Δ), plus 10 Δ should be added to the chart vergence to get the stereovergence. If the stereoscopic arrangement carries 18 Δ, bases out (O. U. 9 Δ), this amount should be added to the chart vergence, and so on for any other prism power.

SUMMARY AND CONCLUSION

In orthoptic training with the stereoscope or stereoscopic arrangement it is desirable that the examiner know at all times the direction of the patient's visual axes, that is, the kind and degree of vergence when the patient fuses corresponding points on the stereogram. The stereoscopic vergence is made up of the plus prism vergence produced by the prisms with the bases out and the chart vergence, which may be zero, plus or minus. The kind and amount of chart vergence is obtained from the simple formula $\Delta = \frac{100d}{L}$, in which Δ equals the number of prism diopters, d the difference in centimeters between the interpupillary distance and the separation of corresponding points on the stereogram and l the distance of the stereogram from the eyes in centimeters. A plus result indicates plus chart vergence, or convergence; a minus result indicates minus chart vergence, or divergence. When the chart is kept at a fixed distance a vergence coefficient is readily obtained, and the calculations may be done mentally. Finally, the resulting stereovergence in prism diopters can be readily converted into meter angles.

O'CONNOR CINCH SHORTENING OPERATION FOR HETEROTROPIA AND HETEROPHORIA

A CRITICAL SURVEY

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It is well known that many procedures that are now standard practices in medicine and surgery were notoriously slow in being adopted and, conversely, that innovations are occasionally widely accepted, only to be quickly discarded by the medical profession.

The O'Connor operation in its original form was described in *The Journal of the American Medical Association* in 1912. The originator modified his technic and reported forty-two operations in 1916. Since that time several additional reports have been made by Dr. O'Connor. Several papers on the subject have appeared, but with the exception of a brief reference in Dr. Luther Peter's book on "The Extra-Ocular Muscles" and a brief description by van der Hoeve in the Graefe-Saemish "Handbuch der gesamten Augenheilkunde," they have all been written by oculists in San Francisco. There has been no widespread interest in the operation, nor has it been adopted by any great number of ophthalmic surgeons. In spite of this, we are convinced from first-hand experience that the operation possesses great merit and will eventually displace all other operations for heterotropia and heterophoria, except in the rare instances in which tenotomy alone in some form is clearly indicated.

We believe that the lack of enthusiasm on the part of ophthalmologists toward the operation is due largely to three principal factors: (1) the difficulty of understanding the operation from published descriptions alone; (2) lack of appropriate instruments for certain steps in the operation; (3) the alleged frequency of untoward reaction of the tissues following its performance.

Read before the Section on Ophthalmology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 16, 1933.

After using the operation in its present form one feels as Dr. O'Connor did in 1930, when he said:

> This method of tendon shortening, which was devised by me in 1911 and first reported in 1912, has mechanical advantages so apparent that I cannot understand the failure of the profession to appreciate them. These advantages are:
>
> 1. The tendon is not cut.
> 2. No tendon sutures are used.
> 3. The shortening is entirely in the tendon itself.
> 4. No change in the shortening can occur until the shortening material is removed (twenty-one days) by which time the tendon loops are firmly united.
> 5. Binocular occlusion is not required.
> 6. Monocular occlusion is not required in many cases
> 7. There is no confinement to bed or house.
> 8. There is no chance for adhesions to form between the site of shortening and the sclera because the eye is not put at rest.
> 9. A maximum shortening equal to the length of the tendon tissue is possible.
> 10. The operation may be done in the office if so desired.

We have been able to substantiate each one of these claims, and it does seem incredible that an operation possessing such manifold advantages has not been universally adopted in the twenty-one years since it was first described.

PREVIOUS DESCRIPTIONS IN THE LITERATURE

Many operators who have read of the operation were undoubtedly loath to attempt it because of the difficulty of visualizing the steps in its performance from the printed descriptions and diagrams alone. When once mastered, the technic seems simple, and yet success with the operation depends on meticulous adherence to certain essential features which require the application of some mechanical skill. In addition to its theoretical advantages, it has proved to be much superior to any other operation with which we are familiar in practice. It permits surgical relief in a group of cases in which prisms fail and which one would not dare attempt to relieve surgically by any other method. No matter what contributions are made to the knowledge of the etiology of heterotropia and heterophoria, the treatment in a large group of cases will necessarily always be surgical.

In reading over Dr. O'Connor's early articles it is obvious that he was not completely emancipated from the influence of the advocates of a tendon-tucking operation, and while he described the running loop operation in his first communication in 1912, it was not until 1920 that he entirely abandoned the idea of a central togue, tucked or sutured to the sclera. While he has demonstrated the method at numerous local and sectional medical meetings, a detailed description of the technic

did not again appear in the literature until 1930.[1] It was described once more in 1931.[2] The only modification of note since that time is the omission of the no. 000 catgut loop about the base of the cinch shortening. To Dr. O'Connor and others who use the operation this loop at first seemed essential to prevent the loops in the tendon slips from retransferring to the "dermal,"[3] but it has been found to be unnecessary and has been abandoned. By omitting this catgut, one possible source of reaction is eliminated, and the slight hump at the site of the shortening, which eventually smooths out in either case, is less apparent in the first few weeks after the operation.

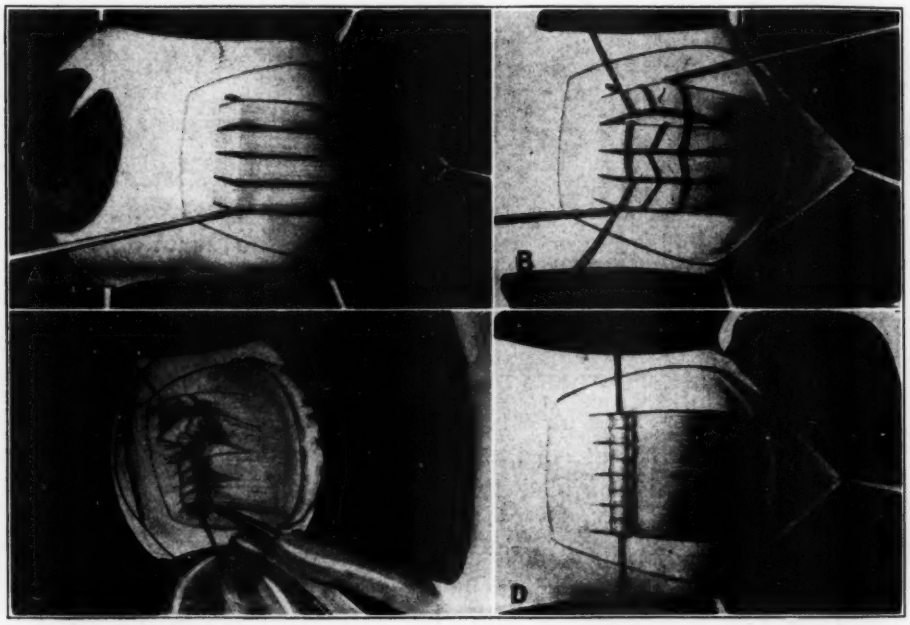

Fig. 1.—*A*, dissection prepared for introduction of dermal suture. *B*, dermal suture threaded around tendon slips, showing double looping as, for example, in moderate heterotropia. *C*, loops being transferred. *D*, transfer completed. The anatomic drawings were made by Mr. G. Ilyin.

It is probably futile to attempt another description of the operation in words. In teaching students we find that more can be accomplished by demonstrating the operation on a model than on the actual patient. The principle can be rendered strikingly apparent by the use of an ordinary comb cleaner made of cotton string to represent the tendon

1. O'Connor, R.: Tr. Pacific Coast Oto-Ophth. Soc., 1930.

2. Barkan, Otto; Barkan, Hans; Randel, H. O., and Smith, H. G.: Squint: Its Physiopathology and Surgical Treatment, Arch. Ophth. **5**:691 (May) 1931.

3. The term "dermal" is a trade name for a particular manufacturer's skin suture. Similar skin sutures are available under other trade names.

and a piece of cord to represent the bundle of dermal. To those with a nautical background, one demonstration is sufficient, but some practice in making the loops is desirable before the operation is attempted. If the dermal is not correctly laid, a transference of the loops to the tendon slips will be impossible. Furthermore, a clean dissection is highly desirable, if not absolutely essential. We have found the accompanying drawings of value in teaching the technic (fig. 1).

INSTRUMENTS

The operation can be performed with the instruments already in the possession of the average oculist. Dr. O'Connor has, however, invented and described two instruments both of which are highly desirable and one of which, the flat, square hook, is almost essential for properly spreading the tendon fibers in order that the tendon slips

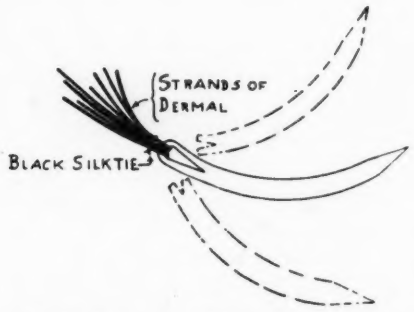

Fig. 2.—Ordinary needle, showing angulation.

may be made of uniform size. The longitudinal splitting of the tendon may be accomplished with a spud or a similar instrument, if care is taken not to wound the sclera, but Dr. O'Connor's hook-shaped splitter is probably superior to anything else for this purpose.

Any short, blunt, curved needle with an eye sufficiently large to carry the multiple strands of no. 000 dermal will work. Dr. O'Connor makes his own needles, but the fact that there are no thoroughly satisfactory needles on the market led us to devise one with a threaded collar of the same caliber as the shank of the needle (fig. 2). The appropriate number of strands of dermal (usually from four to six) are passed through the eye in the usual way, and both free ends of the suture material are passed through the collar, which is then firmly screwed over the eye of the needle. This arrangement makes the diameter of the needle somewhat larger than is desirable, but we have found it more satisfactory than tying the thick bundle of strands together behind the eye of the needle with silk. If the splits in the tendon are made sufficiently long, the multiple suture may be passed around the tendon slips with ease.

Another decided advantage is that the bundle of suture material is held firmly in the direction of the long axis of the needle. This prevents acute angulation (fig. 3) at the point where the dermal passes through the eye of the needle. The angulation which occurred with

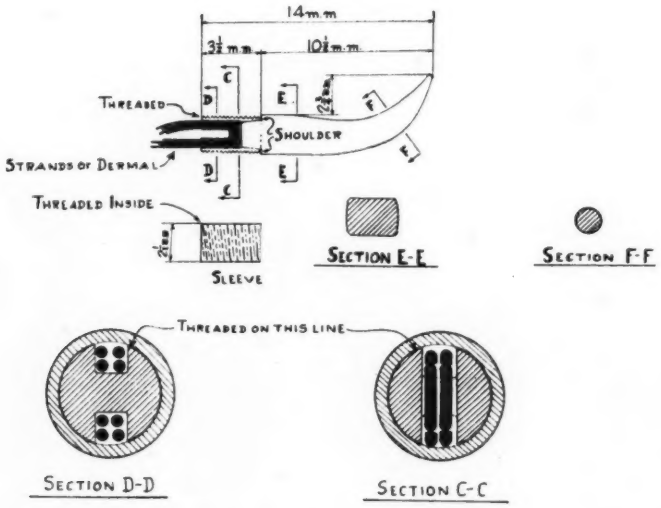

Fig. 3.—Detail of improved needle.

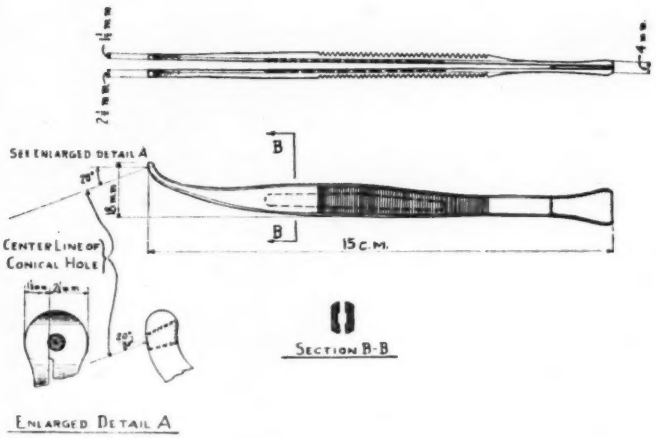

Fig. 4.—Transfer forceps.

the ordinary needle made it difficult to start the dermal through after the needle was passed, slowed up the operation and sometimes required more pull on the delicate tendon slips than was desirable. These annoyances are eliminated by the new type of needle.

In transferring the loops of dermal to the tendon slips no great force is required, but it is sometimes difficult to apply the force at the

right point. Any small curved forceps will serve to pull against, but one with a hole just large enough for the dermal to slip through and with a flat end which can be applied to the side of the tendon is a decided help (fig. 4). The dermal is rendered sufficiently pliable by boiling it for twenty minutes and keeping it in hot water until just before it is used. It should, of course, be cooled to body temperature before it is used.

REACTIONS OF THE TISSUES

Some surgeons have abandoned the operation because of violent reactions of the tissues, ranging from edema with chemosis to actual

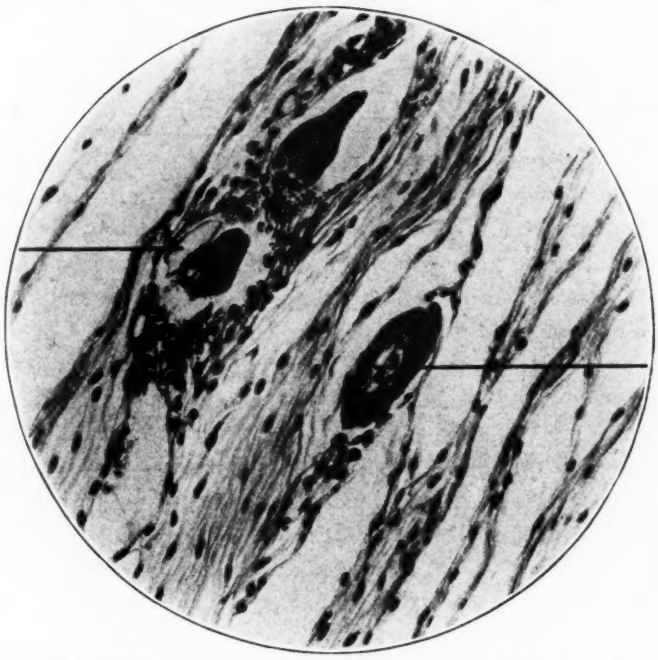

Fig. 5.—Section of rabbit tendon, showing foreign body giant cells containing cotton fibers (black lines).

sloughing. These reactions were at first thought to be due to some chemical irritant contained in the dermal in spite of the fact that prolonged soaking fails to affect its color, and that the dermal is relatively unchanged after remaining in the tissue for three weeks.

The fundamental surgical principle that trauma is to be avoided as far as possible is well illustrated by this operation, and in this sense trauma should, of course, not include merely the maceration and stretching of tissue, but should extend to all forms of mechanical, chemical and thermal, as well as bacterial, insults.

Experimentally, foreign body giant cells have been observed to occur in great numbers about the site of the operation during healing.[2] This

observation was not intended as an argument against the operation, but might be so interpreted by others. Careful search in the centers of these giant cells reveals cotton fibers [4] (fig. 5). This is not surprising, for cotton is widely used in operations on the eye, and even though tightly rolled and moistened it must shred off into the tissues to a degree, particularly if one is operating on a rabbit. This undesirable occurrence, however, will not be more frequent in the cinch shortening operation than in any other operation on the extra-ocular muscles.

Unless constant vigilance is used, compound solution of cresol and alcohol may be introduced on an instrument. The usual corrosive mercuric chloride lavage may often be too highly concentrated, and we have abandoned the use of any solution except physiologic solution of sodium chloride either preceding or during the operation. Solutions of boric acid, which are widely used, are undoubtedly capable of producing unfavorable reactions. The bactericidal power of even the saturated solution is well known to be negligible, but we believe that it is far from harmless when poured on exposed tissue. Some operators have advocated the use of petrolatum or paraffin on the dermal to facilitate its slipping through the tendon loops. We find these substances unnecessary, and we believe on theoretical grounds that their use is undesirable.

Although we have never observed violent reactions in our own cases, we feel obliged to take every precaution against their possible occurrence. It seems only rational that bacterial contamination is the principal cause of reaction. When one is reasonably sure of the sterility of the instruments and suture material, it seems logical to eliminate the possibility of introducing pus-forming organisms from the inadequately sterilized skin of the patient himself. Consequently, we use great care in trimming the lashes so that no cilia or portions of them are left in the conjunctiva or lids. It is wise to trim the cilia with one continuous closure of broad-bladed scissors coated with petrolatum. Lavage of the conjunctiva with a gentle stream of physiologic solution of sodium chloride at body temperature completes the preparation.

The usual gauze covering for the face is, of course, not impervious to moisture and bacteria from the patient's own face. It is therefore advisable to use, as Dr. O'Connor does, a piece of sterilized rubber dam with a hole cut in it for the speculum. The cut edges of the rubber can be arranged in such a way that they completely cover the stumps of the cilia and the margins of the lid. We believe this to be a valuable aid in the prevention of any contact of instruments, sutures and gloves with the imperfectly sterilized skin of the patient.

The cinch shortening operation by itself solves the operative treatment of heterophoria. It is adequate for many hypertropias and for

4. This observation was verified by Prof. Granville Y. Rusk.

TABLE 1.—*Review of Nineteen Cases of Heterotropia*

Patient	Age	Degrees of Arc Before Operation	Refraction	Vision	General Appearance	Result	Comment
			Monocular Convergent Squint				
C. H.	6	20-25	+1.0 sph. +1.0 sph.	1.0 0.4	Straight	6 △ D. exophoria	
H. Q.	19	20	+7.50 sph. = +2.00 cyl., axis 95° +7.00 sph. = +2.50 cyl., axis 90°	0.6 0.3	Straight	Orthophoria	
I. E.	7	20	+2.25 sph. +2.00 sph.	0.2 1.0	Straight	Orthophoria	
R. S.	20	30 4△D. left hyperphoria	+2.00 sph. +1.75 sph. = +0.25 cyl., axis 175°	0.2 1.0	Straight	4 △ D. esophoria 4 △ D. left hyperphoria	Reese resection; aged 7; effect slight
O. D.	6	25	+2.00 sph. = 3° B.D. +2.00 sph. = 3° B.U.	0.4 0.4	Straight	10 esophoria 6 △ D. left hyperphoria	Straight only when vertical deviation is corrected
C. S.	6	30	+2.00 sph. +2.00 sph.	0.5 0.4	Straight	Orthophoria	
R. B.	7	35	+2.00 cyl., axis 150° +0.50 sph. = +2.00 cyl., axis 90°	...	Straight	Orthophoria	
V. S.	7	30	+1.25 sph. Aphacia from trauma	1.0 ?	Straight	.....	
P. C.	4	25-30	+2.50 sph. = +0.87 cyl., axis 90° +2.50 sph. = +0.87 cyl., axis 90°	...	Straight	.....	
M. S.	5	15	+3.25 sph. = +1.00 cyl., axis 105° +2.50 sph. = +2.00 cyl., axis 75°	0.1? 0.5	Out	10 △ D. exotropia	Poor central fixation of right eye
C. P.	10	20	Deformed eye +1.25 sph. = +1.00 cyl., axis 10°	L. P. 1.0	Out	5 △ D. exotropia	Satisfactory cosmetic result
			Alternating Convergent Squint				
L. C.	21	12	+2.25 sph. = +0.75 cyl., axis 180° +2.25 sph. = +0.25 cyl., axis 150°	1.0 1.0	Straight	Orthophoria	
P. E.	7	25	+2.25 sph. +2.25 sph. = 2° B.D.	1.0 1.0	Straight	4 △ D. exophoria 2 △ D. left hyperphoria	
C. V.	3	20	+0.75 sph. = +0.75 cyl., axis 105° +0.75 sph. = +0.75 cyl., axis 75°	0.6 0.6	Straight	Orthophoria	
D. M.	4	25	+1.50 sph. = +0.25 cyl., axis 180° +1.50 sph. = +0.25 cyl., axis 180°	0.8 0.8	Straight	3 △ D. esophoria	
B. D.	13	20	+0.75 sph. +0.75 sph.	0.6 1.0	Straight	12 △ D. esophoria	
F. H.	9	25	+4.00 sph. +3.75 sph. = +0.50 cyl., axis 90°	1.0 1.0	Straight	8 △ D. esophoria	
F. M.	7	15	+0.25 sph. = +0.50 cyl., axis 90° +0.25 sph. = +0.75 cyl., axis 90°	0.5 0.6	Straight	2 △ D. exophoria	Operated on 4 years ago; method unknown
F. H.	6	25	+0.75 sph. = +0.75 cyl., axis 180° +0.75 sph. = +0.75 cyl., axis 180°	1.0 1.0	In	20 △ D. esotropia	Bilateral paresis of superior recti muscles

TABLE 2.—*Results in Four Cases of Heterophoria*

Patient	Age	Deviation Before Operation	Refraction	Vision	Deviation After Operation		Comment
					20 Feet	14 Inches	
			Heterophorias				
P. L.	29	16 △ D. exophoria	−5.25 sph. = −1.00 cyl., axis 88°	1.0	2 △ D. exophoria	5 △ D exophoria	
			−4.00 sph. = −0.37 cyl., axis 105°	1.0			
D. A.	16	40 △ D. exophoria	−7.25 sph. = −2.50 cyl., axis 84°	0.8	10 △ D. exophoria	5 △ D. exophoria	Operation on other eye advised
			−6.75 sph. = −1.00 cyl., axis 90°	0.8			
S. G.	28	2 △ D. exophoria	+1.00 sph. = +0.50 cyl., axis 100°	1.0	2 △ D. exophoria	12 △ D. exophoria	
		10 △ D. right hyperphoria	+0.25 sph. = +0.50 cyl., axis 80°	1.0	1 △ D. left hyperphoria	1 △ D. left hyperphoria	
R. H.	21	22 △ D. exophoria	−3.25 sph. = −2.25 cyl., axis 180°	0.8	Lateral orthophoria	7 △ D. exophoria	Vertical deviation corrected 3/18/32
		26 △ D. right hyperphoria	−6.00 cyl., axis 135° = 1° B.D.	0.5	1 △ D. left hyperphoria	1 △ D. left hyperphoria	Lateral deviation corrected 3/19/32
			Result in One Case of Exotropia from Aphacia				
			Aphacia with Exotropia				
W. W.	43	40 △ D. exotropia	+8.75 sph. = +2.00 cyl., axis 85°	1.0	5 △ D. exotropia	.....	Had never worn glasses; position could be improved but patient was satisfied
			+0.25 sph. = +0.12 cyl., axis 90°	1.0			Patient was satisfied; aphacia from injury 20 years ago

lateral deviations of small degree, but for the correction of large lateral deviations we believe that it should be used in conjunction with a partial central tenotomy, as recommended by Dr. O'Connor in his first paper.

TENOTOMY

Partial tenotomies may be classified anatomically into two types: (1) those in which the lateral borders of the tendon are cut and (2) those in which the borders are undisturbed, the central tenotomy advocated by Stevens in 1883. This type of tenotomy has been criticized as ineffectual, but when used in conjunction with the cinch shortening operation it is far from ineffectual. The reason that it has been considered ineffectual is because it has been used either as the sole surgical procedure for correcting a deviation or in conjunction with an advancement operation which depended for its success on sutures, and all suturing of tendon to the sclera is likely to slip.

Dr. O'Connor has reported one case [5] in which he corrected cyclophoria by the use of Todd's method of lengthening one border of the tendon more than the other, and in a personal communication he called attention to the fact that, when necessary, he operates for a triple effect, that is, lateral, vertical and rotary. If cyclophoria can be cured or relieved by lengthening one border of the tendon more than the other, it is only fair to assume that rotation can and will be produced unless the greatest care is exercised in any form of partial tenotomy if one or both borders are cut. No consideration will, therefore, be given to any type of tenotomy in which the border of the tendon is disturbed as a primary procedure unless rotary effects are desired. All recession operations possess the same grave disadvantage. It seems to us much more logical to rely on a few tendon fibers, perhaps less than 0.5 mm. wide, left in their original anatomic position, than on any amount of scleral suturing, the dangers of which need not be enumerated.

Contrary to Stevens' teaching, we believe that it is desirable to expose the tendon of the short muscle and to spread it on the square hook exactly as in the first step of the cinch shortening operation, and to cut the central fibers off cleanly as near the insertion as possible, then carefully extend the incision to as near the lateral border as seems desirable in any given case.

The arrangement of the spindle-shaped muscle fibers of the extraocular muscles is such that cutting the center of their tendons allows more retraction than cutting the edges. This procedure leaves intact the portions of the tendon that are most concerned with preserving the position of the eye with respect to its rotation about the visual axis

5. O'Connor, R.: Am. J. Ophth. **3**:116 (Feb.) 1920.

and yet allows the greatest alteration in the point of application of the force which the muscle exerts.

Some operators have argued that the function of tenotomy when combined with the cinch shortening is merely to traumatize temporarily and hence place in abeyance the action of the short or overacting opponent of the muscle on which the cinch shortening has been performed. They have said further that it matters but little what type of tenotomy is done, and that the site is unimportant—that even stretching of this muscle will serve as well as a partial tenotomy. We heartily disagree with these teachings. We believe that the site of the partial tenotomy should be as near the attachment of the sclera as possible, and that the tendon and muscle behind the insertion should be traumatized as little as possible. The farther from the insertion nicks or button-holes are made, the more certain is the scar tissue which results to organize, contract and vitiate the effect of the cinch shortening.

If the fibrous insertion is shaved off close to the sclera, the cut portion of the tendon retracts as a unit and reattaches smoothly. This prevents the spreading apart of the more proximal part of the tendon where the fibers are all parallel and if cut splay apart readily.

In the present state of ignorance of the etiology of străbismus and the well known variability of results in what appear to be identical cases in which identical surgical procedures are used, it seems fruitless to attempt to reduce either the amount of shortening or the size of the tenotomy to absolute units of measure because of the other variables in the problem that we have no means of measuring at the present time. Dr. McCool's [6] work on the amount of shortening produced by strands of dermal of various sizes is of great assistance in deciding how many strands to use, but we have no similar guide to the size of the tenotomy. When the combined operation of the cinch shortening and partial tenotomy is done under local anesthesia, it is possible to measure the result with a Maddox rod at the time of operation, and as the immediate result is very close to the final result, one can extend the tenotomy until the proper position is reached, or if an overcorrection has been produced, it is easy to remove enough strands of dermal to allow relaxation to the proper position.

Further, if the degree of strabismus is too great to be corrected by the combined operation of cinch shortening and partial central tenotomy, it is probably better to divide the final result between the two eyes, and after some weeks to operate on the second eye in the same manner. The author of the operation has repeatedly emphasized that more than one operation is likely to be necessary, but we have not found this to be the case often enough to consider it a disadvantage.

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6. McCool, J. L.: Am. J. Ophth. **13**:491 (June) 1930.

Patients are naturally loath to submit to surgical intervention more often than is necessary, and unless the degree of deviation is large, one operation is usually sufficient unless symptoms persist.

It is probably well to begin using the operation for heterotropias, and after some experience has been gained, to attack the even more gratifying field of the heterophorias. Here the cinch shortening operation alone without tenotomy suffices. In these cases fewer strands of smaller dermal (000) may be used, and more splits in the tendon are made. Cases of heterophoria, for example, frequently go undetected, and the patients do not come to us for relief until adult life so that almost all of these operations may be done under local anesthesia, and, again, as the immediate result is very close to the final result, the correction can usually be made in one sitting; the results are extremely gratifying.

SUMMARY

1. The history of the development of the O'Connor cinch shortening operation for heterotropia and heterophoria together with its advantages and present status is outlined.

2. Two new instruments to facilitate its performance are described.

3. Theories in regard to its relation to central tenotomy of the opposing muscle are set forth.

4. Certain important factors in the cause of untoward postoperative reactions are discussed.[7]

ABSTRACT OF DISCUSSION

DR. WILLIAM F. HARDY, St. Louis: The O'Connor operation is twenty-one years old, yet for some reason it has not been given wide approval. It has all the advantages and none of the disadvantages of the tuck. There is less likelihood of torsional effects with the cinch or tuck than with any other, as the natural insertion is preserved. One claim for advancement is that the arc of contact is increased. But does it stay so? Postoperative anatomic investigation shows that the tendon anterior to the original insertion becomes adherent and atrophies, losing the increased arc of contact and resolving itself into an ordinary resection. Those opposed to this statement will say that this will not occur if the operation is done properly. Why there should be any greater need for meticulous care and asepsis in the operation under discussion is not apparent. Care and asepsis constitute a common denominator for all work on muscles. Yet the fact remains that conjunctival reaction seemed disproportionately large in my limited experience with the O'Connor operation, and the continued presence of dermal sutures for twenty-one days appears to me to be a drawback to the procedure. I disagree with Dr. Hosford in the method of producing relaxation. At best we are dealing with variables when working on the extra-ocular muscles. To introduce artificial variables in addition to the natural ones is leaving too much to chance. Numerous

7. For the table of results in twenty-four consecutive cases in which the operation was used, see the Transactions of the Section on Ophthalmology of the American Medical Association, 1933.

operative procedures may be employed to bring eyes to parallelism, but the great opportunity for the cinch technic is in errors of low degree, say from 10 to 20 degrees of deviation, and particularly in the phorias. Except in rare instances, operation on the oblique muscles is contraindicated, and the vertically acting muscles are approached with great caution. If any procedure has a just claim to the vertical muscles it is the O'Connor operation.

Dr. W. D. Horner, San Francisco: We have had the opportunity of doing the O'Connor cinch operation and studying its results for more than three years at the University of California Medical School, where it was introduced by Dr. Joseph McCool. Having viewed it at first with that measure of suspicion which every new procedure bears, Dr. Cordes, my associate, and I have come to adopt it almost as a routine. We believe that it is the best procedure for shortening a muscle. However, as its maximum shortening effect on any one muscle, when used alone, is limited to about 15 degrees, some other procedure must be adopted for lengthening the antagonist if one wishes to correct the average squint. We have satisfactorily combined it, therefore, with recessions, and in a few cases with the Stevens partial tenotomy, as advocated by Dr. Hosford. While the tenotomy alone is ineffectual, it becomes effectual when combined with the O'Connor shortening. We prefer the recession, however, since we believe that we can estimate our effects more closely by this combination. The O'Connor technic is most valuable in cases deviating 12 degrees or less, particularly when this degree remains after a previous attempt at correction, and in the operative treatment of phorias, particularly of the vertical type. The amount of shortening obtained is from about 1 to 1.5 degrees per strand of the medium dermal suture used; that is, with the ordinary technic (four bundles of muscle twice over). We can therefore figure beforehand about how many strands to use in a given case. If an overcorrection is obtained, we can remove one or more strands in the next few days. If we removed all of them we should be no worse off than before operation, since we have done nothing to the insertion of the muscle. Since buried foreign material in any tissue always provokes some reaction, reaction can be expected in this procedure, and for this reason special precautions in technic should be taken to avoid infection. We shave the eyebrow, clip the lashes, and work through a hole in a sheet of rubber dam. The needle and forceps devised by Dr. Hosford are a distinct aid in the technic.

Dr. George N. Hosford, San Francisco: We did not intend to give the impression that it was desirable from the standpoint of physiology to secure the entire result on one eye, although it is more economical. Dr. O'Connor prefers to divide the result between the two eyes if the deviation is more than 20 degrees. The important point is that the operation can safely be done at the early age of 2½ or 3 years. If the eyes are made straight this early there is more likelihood of the spontaneous development of stereoscopic vision. The questions I have most frequently been asked are: How many strands of dermal? How many splits in the muscle? How much tenotomy? These questions cannot be answered in general but the answers must depend on the individual case. The advocate of other operations for strabismus all seem to know exactly how much recession, resection or advancement should be done, though they rarely reduce the quantity to mathematical terms but admit that it depends on clinical judgment. In the present state of knowledge I do not believe that any operation can be reduced to a set of mathematical rules. The procedure is much more like freehand drawing than like mechanical drawing. If the operation is done with a single strand of dermal con-

siderable shortening is produced, but if it is done with two strands of dermal the effect is by no means doubled. However, by increasing the number of strands of dermal up to six or eight the shortening effect is materially increased beyond that which is produced by a single strand. Many surgeons have been able to follow Stevens' technic for partial tenotomy and secure good results. I have not been able to duplicate their results unless under direct observation I have exposed the tendon, isolated the borders and cut the central portion close to the sclera. I had hoped, by presenting this demonstration, to interest some of the beginners in ophthalmology in a procedure which I believe has great merit. In this I have been unsuccessful, but I have been gratified by the interest exhibited by men who have been making ophthalmic history for the past forty years.

PSEUDOXANTHOMA ELASTICUM AND ANGIOID STREAKS

REPORT OF A CASE

M. W. JACOBY, M.D.
CLEVELAND

Recent literature has dealt with the history and etiology of pseudoxanthoma elasticum and angioid streaks of the retina rather extensively, so that details in this respect will be omitted here.

To date seventeen cases of the combined diseases have been reported in the literature, and in no case have photographs of the fundus been presented.[1] Artists' drawings can scarcely give a true reproduction of the pathologic picture.

The case reported here presents the typical clinical picture of each disease, and the ocular changes are verified by stereophotographs.

REPORT OF CASE

Miss W., aged 27, had vision of 6/60 in the right eye and 6/10 in the left eye on July 17, 1933. Four weeks previously the left eyeball was forcibly struck by a tennis ball. About two weeks later central vision became blurred in this eye. Vision in the right eye had been very poor for twenty years.

The patient had "pneumonia" seven times between the ages of 6 and 17 and was seriously ill each time. An operation on the mastoid and tonsillectomy were performed when she was 12, and she had had no sore throats since; she had a sinus infection at the age of 12 with minor recurrences. In January, 1933, she had arthritis which lasted six weeks and was complicated by pyelitis. This was definitely of gonorrheal origin. Her general health at the time of examination was good, though she was underweight.

General examination did not show any focus of infection in the ears, nose and throat. Examination of the chest revealed a suspiciously active pulmonary lesion.

The Wassermann reaction of the blood was negative.

A roentgenogram of all the teeth showed no evidence of infection and no nonvital teeth. Roentgenograms of the sinuses showed no evidence of disease.

External examination of the eyes showed no abnormalities. Ophthalmoscopic examination revealed fine, flocculent, floating vitreous opacities in the left eye; otherwise the media were clear.

In the right eye, adjacent to the temporal side of the disk was a large, densely pigmented, postchoroiditic lesion with an irregular, nonpigmented lesion extending

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From the Ophthalmological Department, St. Luke's Hospital.

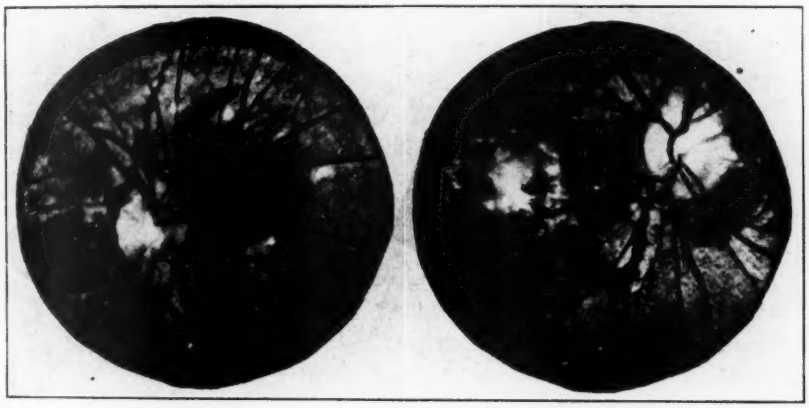
1. Lewis, G. M., and Clayton, M. B.: Pseudoxanthoma Elasticum and Angioid Streaks: A Disease Syndrome with Comments on the Literature and the Report of a Case, Arch. Dermat. & Syph. **28**:546 (Oct.) 1933.

outward to and involving the macula. The area surrounding these lesions presented granular degeneration in the retinal pigment layer. There were many small nonpigmented lesions scattered over the entire fundus.

There were definite, dark angioid streaks radiating outward from the region of the disk, definitely underlying the retinal vessels and not extending beyond the equator.

In the left eye, to the nasal side of the disk, was a large, pigmented postchoroiditic lesion, and over the fundus there were scattered, small, discrete lesions of the same type. There were fresh hemorrhages beneath the retinal fiber layer to the temporal side of the disk, below and temporal to the macula and tiny ones above and nasal to the disk. The macula was very much swollen and occupied by a fresh hemorrhage. The same angioid streaks were present as in the right eye.

The report of the dermatologic examination by Dr. Harold N. Cole and Dr. James R. Driver on August 19 follows: On both sides of the neck, from the jaw down to the clavicle, and extending posteriorly, the skin had somewhat of a yel-



Photographs of the fundus of the right and left eyes taken on Aug. 3, 1933, by Dr. B. J. Wolpaw.

lowish sheen. Closer examination showed numerous linear, slightly raised or puckered, whitish-yellow papular lesions, the size of a pinhead or larger, the skin between them being of a light reddish tint. These linear arrangements followed the somites of the skin. There was definite evidence of atrophy in the lesions. If the skin was lifted with the fingers, it did not fall back in place as quickly as it would normally. It had lost its elasticity. The same type of picture, though more diffuse, was noted especially in the anterior axillary lines of the axillary fossa, and there was a slight yellowish tint to the entire axillary spaces. In this area, the process seemed to be more diffuse. A biopsy specimen was taken from an area on the anterior axillary line on the right, and microscopic examination showed signs typical of senile elastosis.

The patient's condition certainly belongs to the group of the atrophies of the skin. The picture presented here would not justify a diagnosis of any of the other atrophies. There is no question but that the condition is pseudoxanthoma elasticum.

On September 2, Dr. Walter Parker of Detroit examined the patient, and it was his opinion that she had a typical case of angioid streaks in the choroid with pseudoxanthoma.

Treatment with tuberculin following a test was to have been started, but the patient temporarily changed her residence to New York. The treatment is being initiated at the Manhattan Eye and Ear Hospital.

COMMENT

Violent trauma is frequently mentioned as an exciting factor in the disease, though undoubtedly some preexisting pathologic process must be present. In this case definite trauma preceded the acute symptoms in the left eye, though evidence of an old pathologic process was present, and the injury could only have been a destructive factor in the general picture.

Heredity and consanguinity of parents, though having no connection with this case, are often given as etiologic factors. Tuberculosis is also mentioned as a cause. This patient gave a prolonged history of pulmonary infections, and physical examination revealed a suspiciously active pulmonary lesion.

A great variety of opinions are offered concerning the exact pathology of the disease and no pathologic examination has been made in a case previously studied ophthalmoscopically.

Among the principal explanations for the streaks are: (1) fractures in the lamina vitrea or pigment epithelium of the retina (Kofler [2]), (2) pigmentation about a supernumerary system of vessels (von Schrader [3] and Zentmayer [4]) and (3) a wrinkling with folds in the pigment epithelium and in the retina (Alt [5] and Walser [6]).

Pseudoxanthoma and angioid streaks apparently comprise a disease syndrome in which elastic tissue is the site of the disturbance.[1] It is usually seen in the skin, mucous membranes and eyes, but may affect elastic tissue elsewhere, as in the vascular system.

The elastic, outermost layer of the lamina vitrea of the choroid is a mesodermal structure and is accordingly involved in the degenerative changes occurring within the eye. These changes range in degree to extensive destruction of choroidal tissue with associated secondary involvement of the retinal tissues.

Careful examination of the stereophotographs in the case reported reveal some interesting changes. The streaks have every appearance of veins about which there has been an increase of pigment. Their course seems to follow too definite a pattern to be accounted for by fractures of the lamina vitrea. An examination of the superior area of the right eye will definitely disclose a "streak" overlying a retinal artery but

2. Kofler, A.: Arch. f. Augenh. **82**:134, 1917.
3. von Schrader: Ztschr. f. Augenh. **16**:81, 1906.
4. Zentmayer, W.: Ann. Ophth. **18**:472 (Oct.) 1909.
5. Alt, A.: Am. J. Ophth. **26**:171 (June) 1909.
6. Walser, B.: Arch. Ophth. **25**:365, 1895.

having no connection with it. In the large, destructive lesion in the left eye, one "streak" may be seen bridging through the lesion itself. These findings obviously could not be due to a fracture in the lamina vitrea.

Of all bodily structures, the vascular system is perhaps the most capable of modification under altered circumstances. According to Leber, anastomoses between twigs of the choroidal and central vessels around the nerve entrance are in three directions: (1) backward to the pial sheath, (2) straight inward to the region of the lamina cribrosa and (3) forward to the papilla to take part in its vascularization.

With these factors in mind, it seems logical that an altered venous circulation posterior to the equator could readily occur in the choroidal degenerative changes. It is not necessary to depend on the presence of anomalous vessels as a coincidence in such a rare disease as pseudoxanthoma.

SUMMARY

1. A case of pseudoxanthoma elasticum and streaks of the retina is reported with the ocular changes illustrated by stereoscopic photographs.

2. It would appear that trauma may be an exciting factor in exacerbations of an already existing disease.

3. Intercurrent infections elsewhere may be hastening factors in senile elastosis, but they are not necessarily the true etiologic factor.

4. The photographs in this case would seem to establish definitely that the streaks are due to veins which have undergone modification in an altered collateral circulation.

TREATMENT OF MOOREN'S ULCER

F. W. DEAN, M.D.
COUNCIL BLUFFS, IOWA

At a meeting of the Surgical Association of the Rock Island Lines in Colorado Springs, Colo., in September, 1921, I reported a case of Mooren's ulcer.[1] The report was printed in the *Surgical Journal* of July, 1922. It was the report of a case in which both eyes had Mooren's ulcers. The right eye was enucleated to give relief from severe pain, and the left eye was saved with good vision.

Mr. James E. White, aged 70, was first seen on Nov. 9, 1920. Examination revealed corneal ulcers at the outer and the inner limbus of the right eye. Each ulcer was about 6 mm. in length, extending along the limbus, and was about 2 mm. in width at its widest. The conjunctiva was red and thickened for about 3 mm. back from the corneal margin.

For treatment, the eye was cocainized and the entire surface of each ulcer was cauterized with the galvanocautery. An ointment containing atropine in a concentration of 0.5 per cent was put in the eye and a bandage applied. The ulcers began to heal satisfactorily along the edges, with epithelium extending in from the surrounding cornea. In a few days, however, they broke down again.

This process was repeated time after time, though occasionally I used other cauteries, trichloracetic acid, zinc sulphate, iodine, etc. In spite of all, the ulcers progressed and the amount of clear cornea diminished with each cauterization. The ulcers coalesced at the lower part of the cornea.

By the middle of February the clear cornea was limited to a strip about 4 mm. wide, extending from the upper limbus just to the lower edge of the pupil. A smear from the ulcer with Gram's stain showed a mass of pus cells and no organisms. The Ziehl-Neelsen stain was negative for tubercle bacilli.

The right eye was enucleated on February 19. I might be criticized for enucleating the eye before the vision was entirely lost, but I was sure that it would be lost. The patient was suffering intensely, and the left eye was not involved.

The eye was sectioned, and the tissue was examined. Microscopically, frozen sections showed, as the only lesion of consequence beneath the conjunctiva at the base of the cornea, a localized nodular perivascular infiltration by lymphocytes. The conjunctival epithelium immediately over this showed signs of inflammatory hyperplasia with disappearance of the malpighian layer in part, and inward toward the corneal surface, complete ulceration. The conjunctiva and the tissues immediately beneath were abundantly infiltrated by polymorphonuclear leukocytes in the vicinity of the ulcer.

The diagnosis was: chronic subconjunctival inflammation with ulceration. As to cause, there was nothing in the specimen to give even a hint. Tuberculosis and syphilis were possibilities, but the vascular character of the lesion made the former improbable.

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1. Dean, F. W.: Mooren's Ulcer; Report of Case, Surg. J. **28**:165 (July) 1922.

On March 5, two weeks after the enucleation of the right eye, I was again called to see the patient and found an ulcer similar to those which were on the right eye, along the outer limbus of the cornea of the left eye.

I had a specimen of blood taken for the Wassermann test and a guinea-pig inoculated with an emulsion of scrapings from the ulcer. The laboratory reported that the Wassermann reaction was negative. The guinea-pig inoculated with the emulsion of the scrapings from the ulcer of the left eye died, showing gross lesions resembling tuberculosis, but on microscopic examination the lesions were not typical. This condition was therefore probably not tuberculosis.

Fig. 1.—Photomicrograph of a section of the right eye.

Until the laboratory report on the section of the right eye was received the ulcer on the left eye was treated as those on the right had been; that is, by cauterization, atropine and bandage, with the same result. The ulcer began to heal only to break down again in a few days, the reinfection always beginning at the edge adjoining the thickened, inflamed area under the conjunctiva at the limbus. Because of this and because the report from the laboratory on the section of the right eye stated that the only lesion of consequence was beneath the conjunctiva at the base of the cornea, I directed my efforts to the clearing of the inflammation in that area.

On March 18 the eye was cocainized and an incision made through the conjunctiva along the edge of the cornea from the median line above. When near the inflamed part of the conjunctiva, the incision was extended farther out from the cornea so as to pass entirely around the diseased area and again approach the cornea on the lower side of the inflamed area, stopping at the median line below.

All the conjunctiva between the incision and the cornea was dissected off, exposing all of the diseased surface. This diseased area was thoroughly cauterized with the galvanocautery. The burned tissue was curetted away, leaving a bleeding surface. The conjunctiva on the temporal side of the eye was loosened from the eyeball and, with a stitch above and below the conjunctiva, was drawn over and made to cover the raw surface.

The sutures were removed on the fourth day and the conjunctival flap was allowed to retract to the limbus. On the following day, when the flap was off the cornea, I found two small spots which I thought might be a trace of the old trouble. The spots were at the top and bottom of the diseased area. Probably the conjunctival incision did not swing out from the cornea soon enough and was brought in too soon. These two spots were burned with the galvanocautery.

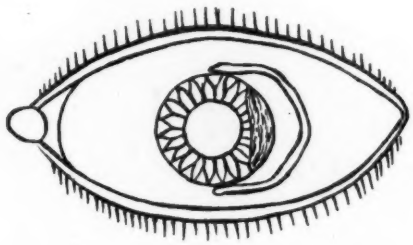

Fig. 2.—Sketch of the incision.

Fig. 3.—Left eye, showing a scar on the temporal side of the cornea.

Healing was prompt and there was never any symptom of a return of the ulceration. The photograph of the left eye was taken on July 18, just four months after the operation.

In recent articles Bedel [2] and Gifford [3] cited reports of Mooren's ulcer in the literature. Neither Bedel nor Gifford found the report of my case, probably because it was not printed in an ophthalmologic journal. Gifford did remember examining a specimen which I sent him.

My experience with the two eyes gave me a definite idea of the treatment which should be followed. I realize my experience is slight but so also is that of others.

2. Bedel, A. J.: Mooren's Ulcer, Am. J. Ophth. **16**:289 (April) 1933.

3. Gifford, Sanford: Rodent or Mooren's Ulcer of the Cornea: Report of Three Cases with Healing, Arch. Ophth. **10**:800 (Dec.) 1933.

Gifford stressed the importance of covering the entire cornea with a conjunctival flap or, if only the surface of the ulcer is covered, making the conjunctiva adhere permanently to it. Any corneal ulcer, after it is sterilized, is benefited by a cover of conjunctiva. But this is not more essential with Mooren's ulcer than with any other extensive corneal ulceration, and an adherent flap is disfiguring. Hypotony is undoubtedly beneficial to healing, and yet the production of an opening into the eye is not without hazards. I do not believe that I could have succeeded with a delimiting keratotomy in my case, because of the extensiveness of the diseased area.

My patient had Mooren's ulcer on each eye. I lost the first eye but learned from the study of that eye that the essential treatment is to remove all the inflamed tissue from the thickened area near the limbus and to remove it thoroughly. The left eye was saved with good vision, as the ulceration was stopped before it reached the pupillary area. The patient lived a number of years with no return of the ulcers.

Clinical Notes

ADJUSTABLE CONTROL STOP TO LIMIT OPENING OF INTRACAPSULAR CATARACT FORCEPS

HAMILTON ROW, M.D., INDIANAPOLIS

The intracapsular forceps described in this article has what I consider to be definite advantages in its construction. The operator can accurately limit the size of the fold of capsule grasped by its blades.

Whether a certain intracapsular forceps should be opened 2 or 4 mm. depends somewhat on individual factors in the technic of the surgeon, such as the following two: (1) the presence or absence of pressure on the globe externally, when obtaining the "bite" with the forceps and (2) the amount of pressure applied to the forceps downward against the capsule as the blades of the forceps are being closed.

If the operator includes between the closed blades of the intracapsular forceps an exceedingly large fold of capsule, he can do this only by increasing the relative tension of the capsule on its contained cortex and nucleus. If the fold of capsule between the blades of the forceps is still larger, the extreme stretching of the capsule about its contents will rupture the capsule during the process of closing the blades of the forceps, or, if not then, as soon as traction is made on the capsule.

To demonstrate the principle involved, if the reader, with the thumb and index finger of his right hand, will lightly pick up a small fold of skin on the back of his left hand, he will find that he has produced relatively little tension of the skin. If, however, he will again pinch up as large a fold of skin on the left hand as possible, great tension will be produced. In these demonstrations, the skin of the left hand is comparable to the capsule of the lens, and the thumb and forefinger of the right hand serve as the intracapsular forceps.

In view of the foregoing, simple demonstrations, it is evident that a certain number of intracapsular extractions must be foredoomed to failure because of uncontrolled, excessive capsular tension produced by the operator's grasping too large a fold of capsule at the time of applying the intracapsular forceps.

It was with these physical principles in mind that an adjustable stop was designed as a control on the Sâfâr intracapsular forceps, to limit the spread of the blades when the forceps was open.

By "setting" this stop, immediately preceding the use of the forceps, any desired amount of opening between the jaws of the blades is obtained. The distance between the blades can be quickly and easily regulated from no opening to as much as 7 mm.

Dr. Sâfâr stated that, in his experience, an opening of 2 mm. between the blades is entirely sufficient. This calculation is substantiated by my limited use of the forceps since designing the stop, which I set to accurately limit the opening between the blades to 2 mm.

Another useful purpose of the stop is to serve as a protection to the blades of the forceps. As stated previously, when the stop is adjusted to its extreme forward position, the blades of the forceps are in apposition, and, when thus adjusted, are protected from damage while the forceps is being sterilized, or while it is stored in the instrument carrying case.

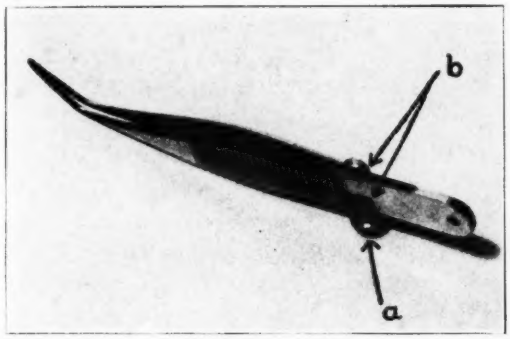

Fig. 1.—The forceps with the adjustable stop (*b*) at its extreme forward position. The lock screw nut (*a*) holds the clamp firmly in position. Note that the blades of the forceps are held in firm apposition protecting the blades from damage while the forceps is being sterilized or when it is stored away in the instrument case.

Fig. 2.—The stop is shown adjusted farther back from the biting end of the forceps. The forceps is ready for use with a 2 mm. opening between the blades. Note that the adjustment screw is on top in this illustration. The stop can be arranged with the screw on the up or on the down side of the forceps.

The same type of stop can be easily made to adapt itself to the Kalt forceps or any other similar, single action intracapsular forceps.

The use of this stop removes one otherwise variable factor from the operator's technic for intracapsular extractions. Such a variable factor not only is distracting, but is the contributing cause in the failure of a certain number of attempted intracapsular extractions.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

OCULAR OBSERVATIONS AND STUDIES OF THE NEW-BORN

WITH A REVIEW OF THE LITERATURE

AMBROSE EARL EDGERTON, M.D.

SAN FRANCISCO

There is no time in life that is filled with more potential danger than the few hours occupied by birth. The fetal head is a tender object, but its structure displays a remarkable combination of qualities which enables it to resist injuries. The brain of the infant is doubtless fitted by nature to withstand without injury a great amount of molding and manipulation. During its passage through the birth canal, the head is subjected to the action of compressive forces and therefore is in a state of stress. At every labor the infant's head is molded a certain amount, the commonest alteration in shape being elongation in a vertical direction and a corresponding shortening in the anteroposterior diameter. This is the result of pressure applied at the opposite ends of the diameter lying between the occiput and the forehead. If alteration in the shape of the head becomes too great, the septums tear because they are nonelastic, and this tearing allows more extreme alteration in the shape of the head and a dangerous disturbance of the intracranial contents. The tentorium is not a vascular structure, and the tearing is not in itself a dangerous injury, but the subsequent changes that may occur in the brain may cause death. Rapid delivery increases the action of these forces and leads to tearing of the tentorium cerebelli and falx cerebri, with a consequent rupture of the vessels. Changes in these septums cause stretching and engorgement of the vein of Galen, leading to rupture of the vein or of its branches.

During the passage of the child through the birth canal, the portion first delivered, usually the head, is relieved from direct pressure by the uterine contractions. Glaser stated that the compression of the undelivered portion of the child by the contracting uterus causes intense intracranial congestion. Intra-ocular engorgement must necessarily

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follow, and often there is retinal bleeding. According to Cushing, the skull of the new-born is well fitted to withstand pressure, by virtue of the fontanels, the easily separated sutures and the medullated soft brain structures within. Any condition which increases the stress of the fetal head in its passage through the birth canal will increase the chances of injury. Contracted or malformed pelves, precipitate or prolonged labor, breech or abnormal presentations, rigid soft parts, forceps extraction and abnormally large heads are factors which predispose to injury at birth. Ocular disturbances due to injury of the brain or its membranes are most important factors. The remote effects from intracranial hemorrhage and trauma often result in ptosis, nystagmus, defective vision from disease of the ocular tract and paralysis of the ocular muscles. Many congenitally blind children have a history of injury to the brain at birth. Schwartz, in 1924, stated that the pathologic processes occurring in the first few weeks of life are completely dominated by injuries of the brain at birth.

CAUSES OF INTRACRANIAL HEMORRHAGE

Tyson and Crawford concluded that long, second stage, precipitous delivery and difficult obstetric procedures are the chief causes of intracranial hemorrhages, and that antepartum health of the mother and hemorrhage of the new-born are not important causative factors.

Ehrenfest claimed that 50 per cent of deaths of stillborn infants and of infants in the first few weeks of life were shown to be due to intracranial injuries seen on postmortem examination, and the same percentage of intracranial hemorrhage was shown in the reports of Womack, Knox, Warwick, Litzenberg and Peterman.

Siegmund maintained that the vascular system is most directly affected by trauma at birth. Mechanical injury to the small vessels causes disturbance in circulation, stasis, diapedesis of erythrocytes and, as a result, impairment of the nourishment of the adjacent tissues.

Hemorrhagic disease of the new-born is considered a contributing cause of intracranial hemorrhage, because it often occurs in various organs of the body in normal labor, and could and probably does occur in the brain, independent of any trauma and without any evidence of clinical symptoms. The amount of hemorrhage is no criterion to serve as an index of the severity of labor or of the amount of trauma incurred.

Daniels stated that hemophilic tendencies in the premature child, its thin skull bones and delicate blood vessels and the occurrence of minor trauma constitute predisposing factors to intracranial hemorrhage. At any rate, the vulnerability of the cranial vessels in premature infants is recognized now as significant in the occurrence of intracranial hemorrhage, and no doubt this is important in the origin of retinal hemorrhage.

Sicherer and Stumpf reported retinal hemorrhage in 32 per cent of premature infants as compared with 18.7 per cent in full-term infants, and Paul found retinal hemorrhage in 40 per cent of premature infants.

There is a certain amount of venous congestion at every birth, and the greater the asphyxia, the greater the congestion, and, secondarily, the greater the amount of minute hemorrhages and effusion of blood into the tissues. The congestive ecchymoses are mostly capillary and are scattered through the various organs and tissues. Most of them are of no pathologic importance, but in the brain and eye they are apt to cause trouble. It is the resulting structural damage that is of the greatest importance. Asphyxia can be caused by pressure on the cord during delivery, by premature separation of the placenta, a diseased placenta and a contracting placental site and by prolonged dry labor and prolongation of the interval between birth of the head and birth of the shoulders. The statistics of Manton and Warwick showed that in approximately 20 per cent of all head births the children entered the world more or less asphyxiated.

Ehrenfest stated that asphyxia is a contributing cause of intracranial and intra-ocular hemorrhage, and Wolff and Buchanan made a similar statement. Sicherer and Stumpf cited a case in which asphyxia was associated with hemorrhage into the vitreous and into the anterior chamber, and they believed that the bleeding was due to venous congestion which depended on the position of the head, because in their series of cases both eyes were not affected in the same degree. No method has been devised to estimate the amount of carbon dioxide in the blood during intra-uterine life, but it is no doubt increased if the oxygen coming from the placenta is decreased.

Maternal deficiency in calcium may be a factor in lessening the coagulating time of the blood. Dietary errors and a deficiency of parathyroid in the blood may also be important factors. Moore and Brodie claimed that a lack of vitamin B in the mother's diet was a contributing cause of hemorrhage in the infant at the time of birth.

Solution of pituitary is given as a cause of intracranial hemorrhage by Ehrenfest, Davis and King, but Stein advised its use in safeguarding the contents of the infant's skull. Hemorrhagic disease, jaundice, hemophilia and instruments have been cited by various authors as causes of intracranial hemorrhage. Intracranial hemorrhage may occur even before birth, and cases have been reported by Gibb, Osler and Seitz.

Most investigators claim that cerebral hemorrhage is more frequent in cases of breech presentation, but Migliaccio found that it occurred in almost as many cases of vertex presentation.

Brissaurd and Dejerine reported that syphilis favored the occurrence of intracranial hemorrhage, while Kearney claimed that syphilis was an etiologic factor in only 1.7 per cent of cases. Bailey stated that in the past too great a proportion of deaths of infants was attributed to syphilis and that it was given too great a prominence as a causative factor. The literature is rather scant concerning ocular lesions in fetal syphilis. Friedenwald examined the eyes of 175 infants who either were still-born or died during the first few months after birth, and 30 showed evidence of congenital syphilis. Microscopic sections of the eyes were made, and 9 of the 30 showed ocular inflammatory lesions.

Injuries to the eyes at birth may be external or internal. Externally, these injuries manifest themselves as subcutaneous hemorrhages, edema, tearing and bruising of the lids, corneal injuries, muscular paralyses, fractures of the orbit with hemorrhage, causing exophthalmos and dislocation and extrusion and avulsion of the eyeball. Internally, hemorrhages may occur in any part of the eyeball as the result of injury of the iris, ciliary body, choroid or retina. The lens may be dislocated or retroverted, and perforation may cause the loss of the eye.

Both intracranial and retinal hemorrhages occur more often than is thought to be the case. It is difficult to estimate with any degree of accuracy what percentage of children have cerebral hemorrhage of one type or another at birth. It is known to be a common cause of infant mortality and is closely linked to ocular hemorrhage. One cannot be considered without the other. There is a marked similarity between the vascular condition which exists in the eye and the cerebral ventricles. In both places, the vessels have on one side a body fluid under a lower degree of pressure than in the vessels.

Injuries at birth cause trauma to the eyes of the child in three ways: by injury to the lids and eyeball, by retinal and intra-ocular hemorrhage and by intracranial injury to the visual tracts and to the nerves supplying the ocular muscles and the associated centers.

The most serious injuries to the eye are the product of direct injury. The bulb may be gouged out, crushed or severely bruised by forceps, by a tip of the blade or by a finger of a careless attendant. Cases have been reported in which the eye socket has been taken for a rigid cervix and forcibly dilated.

CAUSES OF RETINAL HEMORRHAGE

No doubt trauma is the cause of the greatest number of cases of retinal hemorrhage. It can be looked for in all cases of forceps deliveries, breech presentations and difficult and long labors, especially in primiparas if solution of pituitary has been used, and in cases of hemorrhagic diathesis. It is also seen in cases of immature and premature labors, toxemia and asphyxia. Syphilis, too, plays a part, especially in pre-

maturity, by causing weakness in mother and child and fragile blood vessels after an unnatural delivery.

Retinal hemorrhages are frequent in the new-born. Their size varies, and they are most frequently located at the posterior pole. Absorption is rapid, and all traces of bleeding soon disappear. Hemorrhage at the macula may lead to permanent change. Detachment of the retina may occur, and this condition may explain congenital amblyopia.

Retinal hemorrhages at birth probably represent the combined result of abnormal fragility of the walls of the vessels, increased intravascular pressure and often a decreased coagulability of fetal blood.

Many investigators have suggested causes of retinal hemorrhage, since Jaeger made his systematic study of ocular injuries in 1861. Thompson and Buchanan expressed the belief that the hemorrhages are the result of increased blood pressure, due to obstruction of the fetal circulation. Wolff agreed with this theory. Bahn stated that the retinal bleeding had been attributed to diapedesis from the veins, either in the course of a slight asphyxiation or because of the interval which existed between the cessation of the maternal circulation and the full action of the child's heart. Paul believed retinal hemorrhage to be due to loops of cord around the neck. Stumpf later reported retinal hemorrhage in 14 per cent of 63 babies born with loops of cord around the neck. Koenigstein, Knapp and Juler considered this to be a contributing factor. Wolff and Stumpf called attention to asyphxia as a cause of retinal hemorrhage, and the latter found retinal bleeding in 6 of 18 cases of asphyxia, or 33 per cent. Contracted pelves may be a contributing factor, but Stumpf stated that retinal hemorrhage was as frequent in infants delivered normally as in those delivered through contracted pelves, and Eades also thought that contraction played only a secondary part. Paul found that 50 per cent of his new-born infants with retinal hemorrhage were delivered through contracted pelves, while Jacobs found this to be the case in 10 per cent, Eades in 20 per cent, Seissiger in 33 per cent and Hoffman in 50 per cent. Sicherer and Stumpf thought that syphilis played a part, but the finding of as many retinal hemorrhages in nonsyphilitic infants would refute this idea. Jacobs and Eades did not find retinal hemorrhage in any of their syphilitic infants, but the former found it in 41 per cent of infants delivered by forceps, and the latter, in 70 per cent of infants delivered instrumentally.

Signorelli stated, regarding postretinal hemorrhage, that as there is a general increase of intracranial pressure, and as the vessels of the retina do not have as substantial a background as the other blood vessels of the brain, hemorrhage will possibly take place at this particular site and so relieve the intracranial pressure to a certain extent, thereby

preventing further oozing or further damage, unless the condition is an unaccountable one, such as hemophilia.

Ehrenfest stated that severe intracranial pressure at the time of birth affects the eye through hypertension in the supravaginal space about the nerve and through impeded venous return of the retinal circulation, which drains into the cavernous sinus. Metzger emphasized the importance of a negative pressure in the skull during delivery as the genesis of retinal hemorrhage.

Koenigstein considered that these hemorrhages arise during the first act of respiration and are not due to pressure at birth.

In 1924, Coburn reported the results of his examinations of the eyes of 37 infants, who either were stillborn or died a few days after birth. Most of the hemorrhages were found in the equatorial part of the eye up to the ora serrata, while the posterior portion, especially the macular region, presented fewer extravasations. Detachment of the retina in the papillomacular fold was generally observed. Four fifths of the eyes examined showed engorged vessels, even when there had been no extravasations. The retina was the chief site of the extravasation in the 17 cases in which hemorrhage was found. Hemorrhages were most frequently found in the inner layers of the retina, especially in the nerve fiber layer. The subhyaloid hemorrhages were usually massive and occurred as the result of rupture of the blood vessels lying close to, or on the inner surface of, the retina. Coburn believed that this was another proof of the theory of rupture, as large cavities entirely filled with blood were seen in which no signs of retinal elements were observed. The frequency of retinal hemorrhage is remarkable when contrasted with the infrequency of choroidal hemorrhage and the vascularity of the membrane. Coburn suggested as a cause of retinal hemorrhage a disturbance of the retinal circulation from compression of the optic nerve and its central blood vessels. This he claimed was due to distention of the optic nerve sheath with cerebrospinal fluid from compression of the head during birth. This same fluid returns to the cerebral lymph spaces after birth, leaving practically no trace of its former presence, except its secondary effect in producing retinal hemorrhage. The occasional finding of a few blood vessels stranded in the sheath of the optic nerve is partial evidence of the truth of this statement.

No doubt pathologic studies of the eye in infants would reveal a much larger percentage of retinal hemorrhage than is seen by an ophthalmoscopic study of the fundus. Most investigators believe that retinal hemorrhages are due to intense congestion of certain vessels during labor, unless there is a direct injury to the eye. Schleich's theory that the compression of the cavernous sinus interferes with the normal emptying of the ophthalmic vein is supported by Sicherer. A further

support of this theory is the fact that in the more common left occipital anterior presentation, hemorrhage is more likely to occur in the right eye or, if bilateral, it is more pronounced in the right eye. The opposite results are found in right occipital anterior presentation. Schleich and Sicherer believed that the congestion in the eye develops only secondary to intracranial pressure and that the ocular lesion is more often found on the side on which the sinus is exposed to greater pressure. Davis, however, stated that in the majority of labors the left side of the face is directed toward the promontory of the sacrum, and as no pelvis is absolutely normal in contour, pressure is apt to develop in this region, and thus the left eye may be injured. Juler, however, did not find retinal hemorrhage more common in the right eye following left occipital anterior presentation or the reverse in right occipital anterior presentation.

Naumoff believed that during labor, while the head is being molded, the spinal fluid is forced into the subvaginal space of the optic nerve, causing a strangulation of the vena centralis retinae. Otherwise, Naumoff stated, choroidal hemorrhage would occur as often as retinal hemorrhage, and choroidal hemorrhage at birth is rare. Retinal hemorrhage was seen in a few cases in which the subvaginal space (intersheath) was entirely absent.

Schleich and Sicherer believed that the hemorrhages from the smaller retinal vessels are the result of congestion, and that etiologically they have the same relation to the minute and larger cerebral hemorrhage. These varied results may be due to the difference in time of the examination. Small hemorrhages are absorbed quickly, and delayed examination may not show any disturbance in the fundus. Apparently these hemorrhages cause no trouble unless the macula is involved, and this may be the cause of amblyopia ex anopsia, even though no lesion can be seen in the fundus. Jacobs, however, differs with this view and considers them of little importance.

No doubt hemorrhages occurring from injury to the eyes are followed by complete recovery, but it seems probable that in some cases there is permanent damage to vision. Thompson stated that the possibilities of damage to vision from hemorrhage, whether fundal, orbital or intracranial, are very numerous. These hemorrhages are no doubt a normal result of labor, and the more difficulty encountered at delivery, the greater is the tendency to retinal hemorrhage. A large number of normal children are born with retinal hemorrhage.

Juler, discussing retinal hemorrhage at birth, stated that between 10 and 20 per cent of new-born infants show retinal bleeding. This, he thought, is probably due to congestion of the retinal veins in the cavernous sinus which are subjected to great pressure during labor. He

concluded that abnormalities of labor do not have any effect on the incidence of retinal hemorrhage, and Jacobs reported that presentation has no definite relationship to retinal bleeding. Bedell, Jacobs and Juler each described a case of retinal hemorrhage in an infant delivered by cesarean section. Cameron stated that in cases of subdural hemorrhage the ophthalmoscope showed retinal hemorrhage in 60 per cent of cases, and that edema of the disk appeared in some cases. Rowland found 194 hazy disks in the examination of 400 babies.

Sicherer and Stumpf and Koenigstein believed that the blue-gray color of the disk which is sometimes seen immediately after labor is due to a hemorrhage into the subvaginal space of the dural sheath but that in a few days this blood absorbs and the color of the disk returns to normal.

Choked disk is very rare in injuries to the brain. It occurs in less than 1 per cent of adults, and no doubt it is just as rare in the newborn. It is usually seen in persons suffering from extradural hemorrhage, and yet it has to be quite extensive to precipitate an early medullary edema and death. In Sharpe's series of cases no infant with intracranial pressure showed choked disk. Grulee stated that in a series of cases in which intracranial hemorrhage was suspected, careful observation of the disk failed to reveal any change that could be determined as peculiar to intracranial hemorrhage or to an increase of intracranial pressure. Matzinger, however, stated that choked disk is one of the most reliable and important signs indicative of cerebral hemorrhage following delivery, and Kearney found papilledema in cases of intracranial hemorrhage.

SEVERE EYE INJURIES NOTED IN THE LITERATURE

Whenever forceps are used, trauma of some kind may be expected. The lids are often injured by a blade of the forceps. Hematomas, lacerations and ecchymoses may occur. Würdemann cited a case in which the eyelid was torn away in a delivery by a midwife. Similar cases have been reported by Nordquist, Wolff, de Schweinitz, Thompson and Buchanan, Truc and Gad. Contractures of the palpebral fissure due to trauma of the sympathetic nerve fibers, ptosis of the upper lid due to paralysis of the oculomotor nerve and lacerations of the lacrimal duct have been reported. Several cases of exophthalmos, nystagmus, muscular paralysis, luxation and avulsion of the eyeball are on record, as well as cases of crushing of the eyeball, luxation of the lens and corneal opacities, corneal rupture, retinal hemorrhages, cataracts, strabismus and hyperopic astigmatism caused by instruments and noninstrumental delivery. Fractures of the skull may involve an injury to the optic nerve and other nerves in their course through the optic foramina or fissures to the eyeball. Berlin stated that these fractures occurred

perpendicular to the line of force, and that the compression was due partly to the forceps and partly to uterine pressure.

Several cases of atrophy of the optic nerve after birth injury have been recorded by Mackenzie, Naumoff, Sidler-Huguenin, Muhsam, Loppen, Nordquist, Pihl and Fischer. Buchanan said this was usually due to orbital collapse which caused cutting of the orbital muscles and resultant hemorrhage into the optic nerve or its sheath with a defect in vision in later life. Wolff and Goldwasser collected reports from the literature of 244 cases of serious injury to the eye, among which were 28 cases of orbital fracture and 30 cases of exophthalmos, all of which ended fatally. Berkeley stated that fracture of the skull or laceration of the tentorium might produce intracranial hemorrhage followed by ptosis of the lids, paralysis of the external rectus muscle or a combined paralysis of the sixth and seventh nerves. Truc stated that the fractures are usually of the frontal portion of the orbit owing to depression by forceps and that death from cerebral hemorrhage usually occurs. In some cases the eyeball has been partially dislocated; the optic nerve is stretched and pulled out of its normal position and the fibers are damaged, leaving the retinal vessels undamaged but attached over the edge of the cavity left by the dislocated nerve.

Schroeder, Bloch and von Hoffman believed that hemorrhage occurs into the meninges following fracture of the frontal bone, and that this might account for some of the cases of atrophy of the optic nerve and paralysis of the ocular muscles seen later in life.

It is certain that injury at birth must at times result in pathologic changes in the eye, whether or not instruments have been used, and that any part of the eye may be involved. Many abnormal conditions have been reported. Cases are on record of avulsion of the eye which may have been caused by a blade of the forceps or by a piece of fractured bone from the orbit. Just to illustrate what may happen to an eye in the process of birth, Gifford cited the most remarkable case of Hoffman's. Hoffman stated that he

> . . . was called to a woman, who had just given birth to a child and found the infant's right eye hanging out on the cheek, attached only by the inferior rectus and some loose connective tissue. The birth was spontaneous but protracted. Three years later he was called to the same woman and on account of delayed birth and failing uterine power, delivered the child with forceps without difficulty. As the head was delivered, the right eye of the infant fell into his hands. It was quite certain that the forceps had played no part in enucleation of the eye, but the frontal bone was much depressed. As the mother died after a third delivery, a section was made which showed the conjugate diameter to be only three inches. The junction of the last lumbar vertebrae with the sacrum was not rounded off in the normal manner but presented a sharp angle.

This is a vivid example of how nature can accomplish an enucleation at the time of birth. Other examples are reported by de Wecker,

Bock, Turnbull and Polliot. Goldwasser reported a similar case in which the eye was finally found in the placenta. De Wecker described a case in which the orbit, in a face presentation, was mistaken for the anus in a breech presentation and the eyeball was gouged out by the finger. Bock and Redemans each reported a similar case. Maygrier and Zangarol each described a case in which the eyeball was found after birth in the child's linen.

Lomer reported a case of forceps delivery which caused palpebral edema and exophthalmos and resulted in the death of the infant. Post-mortem examination showed fracture of the roof of the orbit and retro-bulbar extravasation of blood. Lomer referred to 4 similar cases, and Beaumont, Reese and Bouchut each reported a case. Zweifel, in 1895, and Shukowski, in 1902, each reported a case of orbital fracture with total protrusion of the right globe from the socket. The blade of the forceps had lain diagonally over the right frontal bone. Similar cases have been described by Servel, Haumer, Gad, Peck and Philipsen. In 1907, Bruno Wolff reported 4 cases of asphyxia, exophthalmos and fracture of the orbits due to instrumental delivery in 581 cases of labor. There are 7 similar cases in the literature, a case each having been reported by Danyan, Pajot, Dittrich, Ahlfeld, Coccius, Hoffman and Fritsch. Borland, Veasey and Crawford each described a case of bilateral exophthalmos following a natural labor. Wengel stated that outside of forceps, a narrow pelvis predisposed, more than any other factor, to obstetric trauma, and that forcing of the head into a narrow cavity predisposed to luxation of the globe. Thompson and Buchanan described a case in which the right eye was lying on the cheek held by the optic nerve. Steinheim, Maygrier, Snell and Donaldson each reported similar cases.

Wintersteiner, in 1899, described a case in which the right globe was crushed. There was also a fracture of the ethmoidal bones, the infant having died from severe epistaxis. In this case the anterior and posterior chambers were filled with blood. Section showed extensive hemorrhage between the ciliary body and sclerotic. Lomer, Zacke and Leopold have reported similar cases.

Friedenwald, in 1918, reported a case of difficult labor and forceps delivery in which the left eyeball was completely luxated and the eyelids were closed behind the eye. Efforts to reduce the eyeball were unsuccessful until an incision was made at the outer canthus. Two years later, the eye was examined and found to be normal in every respect. The author reviewed the literature on 29 similar cases.

Peters, in 1907, reported a case of forceps delivery in which the mastoid was compressed and a tear extended into the facial canal with paralysis of the facial nerve. Peck, in 1898, described a case of instrumental delivery in which there were marked subconjunctival bleeding,

swelling of the lids and flattening of the globe, from which a traumatic cataract developed. Two more such cases were reported by Posey. The case described by Schmidt-Rimpler showed a choroidal tear, which he claimed was due to instruments. De Beck cited an interesting case of optic atrophy and fracture of the optic foramen due to pressure by the forceps.

Many cases of right-sided and left-sided facial paralysis due to injury by forceps have been reported in the literature. It is estimated by Ehrenfest that paralysis of the facial nerve occurs in 10 per cent of forceps extractions. Jacobs agreed with this percentage, and Gans found it in 14 per cent.

Wengel stated that cerebral hemorrhage occurs often in spontaneous delivery, and that paralysis of the ocular muscles due to this cause is not unusual. Knapp, however, claimed that paralysis of the ocular muscles during labor is rare, and that the facial nerve is the most frequently affected, usually temporarily. Capper found strabismus in 3 per cent of his series, and there were twice as many convergent as divergent cases. Ehrenfest believed that strabismus is usually seen in cases of intracranial hemorrhage, owing to effused blood irritating the cortical center of coordinate ocular movements in the angular gyrus. Several investigators, including Nettleship, Borland, Allison, Machaffie, Sidbury, Peterman, Gasteiger and Sidler-Huguenin made similar reports.

Scrini reported 60 cases of alternating squint in 136 new-born babies following internal injury of the orbit or of the brain. In his cases, it occurred most frequently in primiparas and during prolonged labor.

Gifford stated that congenital paralysis of the internal or external rectus muscle is due to pressure at birth and is not apt to be due to nuclear or intracranial lesions, intra-uterine inflammation or muscular aphasia, although many such cases have been described. He reported 194 cases from the literature and from his own series of cases.

According to Panas, in forceps delivery the sixth nerve is liable to injury where it crosses the petrous bone and especially when the bone has suffered lateral compression. Bloch examined 438 cases of paralysis of the sixth nerve and classified 31 as congenital because the defect had existed since birth. He gave three causes that would account for paralysis of the external rectus muscle: laceration of the muscle by instruments, injury of the sixth nerve in the orbit and intracranial injury affecting the nucleus. His investigations covered a period of twenty years, and during this time he had examined 116,000 patients.

Moebius thought that congenital squint is due to atrophy and disappearance of the nuclei, and Kuhn believed it to be due to primary nuclear aphasia. Küstner stated that central paralysis of the ocular muscles is caused by bleeding in the base of the brain, due to fracture of the skull during delivery. This was substantiated by Peters.

Ophthalmoplegia, total or partial, may be congenital. The abducens and the inferior rectus muscle are most often involved. The superior rectus muscle may be absent, especially in cases of congenital ptosis. These anomalies are due to intra-uterine errors in development by cleavage from the embryonic mass. Cases of congenital ophthalmoplegia have been reported by Motolese and Schenke.

Reyer and Lowry, in 1906, reported a case of paralysis of all muscles of the eye except the external rectus, following prolonged labor. Uhthoff described a case of paralysis of the third nerve, which he thought was due to a nuclear lesion. Similar cases have been reported by Crouzon, Posey, Truc, Valude, de Wecker, Pajot, Eason and Franceschetti.

Juler referred to a case of unilateral myopia of 7 diopters associated with damage to the lids and parietal bone on the same side as the myopic eye. Thompson, Ormond and Shumway each cited a similar case, and Collins reported 3 others.

Thompson and Buchanan described a case in which there was retroversion of the ocular contents following severe pressure on the head of the infant during birth. The pars ciliaris retinae was almost stripped free from the ciliary body, and the lens was found lying against the entrance of the optic nerve.

Almost any kind of disorganization of the eyeball may occur, it seems, from pressure at birth even though the eyeball is not ruptured, and yet the eye can stand a great deal of trauma, as is evidenced by cases that show a rupture of Descemet's membrane. The cornea usually escapes injury, but trauma following the use of forceps has caused corneal opacities and tears of Descemet's membrane. The latter condition was first reported by Haab in 1891.

Thompson and Ballantyne, in 1903, stated that corneal lesions at birth may result in temporary or permanent opacity. The temporary opacities, which are of comparatively frequent occurrence, are due to edema. In the cases with permanent opacities, there is a rupture of Descemet's membrane and sometimes of the fibrous lamellae adjoining it, with the formation of cicatricial tissue which remains as a scar. The rupture is generally linear and vertical, but it may be horizontal or eccentric. It is undoubtedly caused by direct pressure on the eyeball by the instrument. Following rupture, there is edema of the cornea, and often some inflammatory reaction occurs. They reported having seen 8 cases of traumatic or linear keratitis, in 1 of which there was hemorrhage in the anterior chamber at birth and in 1, optic neuritis with severed ocular muscles. These injuries were all due to forceps. Similar cases have been reported by Brav, Peters, Nagel, Jardine, Sidler-Huguenin, de Wecker, Buchanan, Collins, Noyes, Fejér, Dujardin,

Cargill, Truc, James, Stephenson, Chance, Bedell, Blaaw, Gratiot, Rupprecht, Heesch, Peck, Wengel and Green.

Von Hippel stated that rupture of the posterior elastic lamina occurred without laceration of the corneal substance. He claimed it was due to a sudden increase of intra-ocular pressure during birth. Ballantyne examined 3 cases with the slit lamp, and tags of Descemet's membrane were seen extending into the anterior chamber. The tears appeared as glassy ridges which often extended into the anterior chamber. They were highly refractile and were usually vertically placed. In all, there was a definite clouding of the cornea.

Corneal trauma at birth must be differentiated from opacities due to keratitis in utero, anomalies of the cornea due to faulty embryonic development, hydrophthalmos and superficial keratitis.

Congenital anterior staphyloma was first described by Beers in 1813, and up to the present time about 30 cases have been reported by Sonnenmayer, Crampton, Steinheim, Freital, Runte, Pincus, Westhoff, Nettleton, Collins, Stock, de Schweinitz, Richter and Gallenga.

Parsons described a case which on section showed Bowman's membrane to be absent. Descemet's membrane was intact on each side for a distance of 1 mm., and the epithelial layer only extended 2 mm. from the limbus. The iris was reduced to distorted pigment epithelium. The ciliary body, vitreous, retina, choroid and optic nerve were infiltrated with leukocytes. He believed that infection of the eye was from the amniotic fluid, and that endogenous infection was improbable.

In regard to inflammation of the eyes at birth, it cannot be said that they are free from any single infection. Many cases have been reported of gonococcus, pneumococcus, staphylococcus, colon bacillus and Koch-Weeks bacillus infection, as well as of a conjunctivitis due to powder, talcum and epidermal débris.

Iritis in the new-born is usually due to syphilis. Tuberculous iritis practically never appears before 2 years of age, although Meyer reported a case in 1930. True adhesions of the iris to the anterior capsule of the lens are occasionally found, and persistent pupillary membrane varies from scarcely visible filaments to dense bands that cross the pupil and are sometimes attached to the posterior surface of the cornea.

Cases of buphthalmos and hydrophthalmos have been reported, the latter associated with Recklinghausen's disease. Congenital glaucoma can usually be accounted for by some defect in the development of the ligamentum pectinatum iridis or canal of Schlemm. One or both have been found to be absent.

Lachman, in 1927, reported a case of bilateral hemorrhagic detachment of the retina in a new-born child. There are 12 other cases in the literature described by Leber, Sattler, Santos, Fernández, Clarke, Stoewer and Rockliffe.

Blue sclerotics at birth were first described by von Ammon in 1841. He noted that the sclera was very thin, and he believed that the blue appearance was due to transparency of its coat. Since that time cases have been reported by Harman, Bolten, Peters, Kunii and Buchanan.

Dacryocystitis and obstructions of the tear sac usually go together, and may be due to bacteria, epithelial casts, exostoses or anomalies of development. Many cases of this condition have been reported in the literature by Santos Fernández, Pechin, Terson, Fage, Cassimatis, Kin, Hirsch and Buchanan.

Spontaneous nystagmus and the intensity of deformation of the skull were investigated in 100 infants by Stern and Schwartz. They found the greater number of cases were due to hemorrhage, caused by the difference between high intra-uterine and low external pressure. The condition was present in one third of the 44 normally delivered infants and in all but 11 with faulty presentation.

Under degenerations have been reported cases of retinitis pigmentosa, ocular palsies, postnatal cataract, symmetrical macular pigmentary degeneration, amaurotic family idiocy, Leber's optic atrophy, Doyne's family choroiditis and nodular degeneration of the cornea.

Many anomalies of development may occur in the optic apparatus of the new-born, and many commonly called congenital are due to injury at the time of labor. As a rule, developmental defects are more apt to occur in the highly specialized structures.

Microphthalmus, cryptophthalmus, cyclopia, polycoria, corectopia and coloboma of the iris, lid, lens, disk and choroid are all congenital defects, as are aniridia, nevus, persistent pupillary membrane, glioma, persistent hyaloid artery, cataract, opaque nerve fibers, posterior cortex veins, ectropion, entropion and pseudoneuritis. Various other anomalies have been reported, such as dermoids, cysts, angioma, cephalcocele, skin bridges from the lids to the globe, congenital dislocation of the lens and abnormal insertion, bifurcation, absence or aplasia of the nerve.

Coloboma of the iris may occur in any position and may be multiple, according to Ida Mann. Many cases of coloboma of the retina and choroid have been reported. The colobomas are usually below and extend nearly to the papilla.

There are about 20 cases of atypical coloboma of the choroid in the literature, and of this number 9 were in the upward direction and were reported by Johnson, Rindfleisch, Derby, von Hippel, Gifford, Mann, Clausen and Finlay.

Cases of unilateral and bilateral aniridia at birth have been described by Gallois and d'Autrevaux, Gifford, Buchanan, Collins, Smith, Harbridge, Lawson, Mayou, Stephenson, Gil and Locatelli Rivas.

CONCLUSIONS

In my series, 458 infants were studied shortly after birth to observe and record the intra-ocular and extra-ocular conditions. Most of the infants were examined on the first day, a few hours after birth. The anterior segment was examined with a plus 20 diopter lens in the ophthalmoscope, and a plus 8 diopter lens was used for the lens and vitreous; this was followed by a study of the fundus. A 1 per cent solution of homatropine was used three times in each eye before the examination was made. A pacifier was used in a few cases and was found to be helpful. In manipulation, after the first spasm of the orbicularis ceased, the examination as a rule could be carried out with little difficulty. Sometimes the position of the child's head was changed, and this seemed to allow of orbital relaxation.

At birth the eyes of the child are tightly closed, the skin being thrown into wrinkles and covered with a fatty material from the meibomian glands and cellular débris. The margins of the lids are usually in contact with one another but sometimes there is overlapping. The palpebral aperature is smaller than in the adult. The blood vessels of the conjunctiva are always congested. Mayou stated that in the secretion of the meibomian glands on the third day after birth he had found xerosis bacilli which he thought were derived from the vagina and were almost identical with the pseudodiphtheritic bacillus found there. He believed that the greasy secretion of the margins of the lids helped to prevent the infected vaginal discharge from getting into the conjunctival sac, but no doubt the rapid delivery through the vagina is a more important factor.

The eyes of a new-born infant appear particularly wide open because the longitudinal diameter is much less than in the adult, while the vertical diameter is about the same. In a child the palpebral fissure is about twice as long as it is wide. As a rule, only a little of the sclera is visible, and the palpebral fissure seems to be almost filled by the cornea.

The conjunctiva and the corneal epithelium in the new-born do not offer the same degree of resistance to infection as in the adult. The corneal epithelium is much thinner and the substantia propria is thicker, in comparison with the adult cornea. At birth, there is no lymphoid tissue in the conjunctiva, but lymphoid cells begin to appear around the vessels of the superficial layers in the upper fornices several days after birth. These cells gradually increase during the first few weeks of life until the lymphoid layer is fully formed. The eyeball of the new-born infant is spherical, there being little difference between the vertical and sagittal diameters. Peters stated that the radius of curvature of the infantile cornea varies between 7 and 7.07 mm., and that there were

individual differences in the thickness of the cornea; that the sclera showed variation in its thickness, especially at the posterior pole; that the choroid was distinguished by its poverty of pigment; that the fovea was flat and no reflex was present; that the retina was thinner than in the adult, and that the optic papilla was of a lighter color and 0.1 mm. narrower than in the developed eyes.

Ecchymosis of the eyelids, with or without breaking of the skin, is of frequent occurrence and is often accompanied by hemorrhage of the conjunctiva. Proptosis often occurs, which is due to hemorrhage behind the eye or fracture of the orbital walls. The blades of the instrument may press directly against the eyeball and the orbits may become fractured or collapsed owing to pressure on the head of the infant. Spicules of bone may thus enter the eyeball or pierce the muscles, or the nerve may be severed. The ocular muscles may become seriously injured by pressure, and rupture may occur, as strabismus is sometimes seen shortly after birth and is usually due to injury. Hemorrhage in the cranial cavity, occurring either under the meninges or intracerebrally, may seriously affect the visual tracts. The cornea may be injured by direct pressure or by increased intra-ocular hemorrhage. This may be serious as far as vision is concerned, owing to rupture of Descemet's membrane, as linear opacities usually develop and high astigmatism is apt to follow. The iris may be injured by pressure, and hemorrhage into the anterior chamber may result. This is usually accompanied by more severe injuries, and its occurrence is often of grave prognostic significance. Not all accidents, however, have been due to forceps. The more frequent cases have occurred following instrumental delivery, but the most severe ones have occurred when no instruments were used, especially in cases in which the mother had a marked sacral promontory with malformed pelves. The injuries include avulsion of the globe, fracture of the orbit, facial palsy and lagophthalmos, exophthalmos, opacity of the cornea, rupture of the cornea, paralysis of the sixth nerve and of the superior and inferior rectus muscles. Some of these palsies, however, are no doubt congenital because the muscle has been found to be deficient in development.

In this series hemorrhage into the vitreous and into the anterior chamber occurred in 2 cases; nystagmus was noted 89 times, and facial paralysis was seen twice. In one, an opaque cornea resulting from injury by forceps was normal again in ten days. One case showed lenticular changes, and 8 cases of strabismus were seen. There was 1 case of bilateral coloboma of the iris and choroid.

The iris was always blue or gray-blue and showed a marked development of blood vessels in every case. Seissiger claimed this was due to lack of development of pigment cells in the stroma of the iris so that

the vessels were more apparent. Pupillary membrane was noted in 27 cases. These remnants stretched from the pupillary border of the iris to the opposite side. Seissiger found many remnants of pupillary membrane lying loose in the anterior chamber immediately after birth. Collins and Mayou stated that threadlike remnants and minute stars of pupillary membrane could be found in all eyes by the use of the slit lamp. Koenigstein found pupillary membrane 21 times in 3,000 examinations, and Schleich found it 13 times in 150 cases. By examination with the slit lamp Seissiger found pupillary membrane in 95 per cent of cases and lenticular opacities in 6.6 per cent. He stated that in his series the pupillary membrane absorbed in about three weeks, and there was absorption of the fine network of vessels in the iris. Vogt claimed that embryonal cataract occurred in 25 per cent of cases, and that it was stationary and did not interfere with vision.

The average size of the pupil was found to be from 2 to 4 mm. in diameter. These figures are very close to those of Bartels in 1905. The average horizontal diameter of the cornea was found to be 9.5 mm. and that of the males averaged about the same as that of the females. Kaiser found the average to be somewhat smaller, 9.44 mm., and the variation in size of the pupil averaged 2.6 mm. At birth the pupils respond to light, but in many cases the reaction was not found to be very acute. Peters stated that only the power to distinguish light from darkness exists in the new-born infant. The photophobia present at birth diminishes after the first few weeks of life.

As far as the weight of the children is concerned, retinal hemorrhage was found more often in the heavier infants. Sicherer and Stumpf and Juler found more cases of retinal hemorrhage in the lighter children.

In this series of infants, 238 were males and 220 were females. Retinal hemorrhage was found in males 74 times and in females 57 times. Stumpf contended that a greater percentage of retinal hemorrhage would be found in males because their cephalic measurements were greater and they were therefore subjected to greater pressure. Eades, however, found retinal hemorrhage more often in females.

Blurring of the disks was seen in only 6 cases. Rowland found this condition in 48 per cent in his series of 400 examinations. Eades found a much smaller percentage in his series.

Gould and Kirkendall stated that in their opinion every child was born hyperopic. Fuch claimed, however, that 5 per cent of new-born children were myopic at birth. Jackson stated that more than 99 per cent are hyperopic at birth. In the present series of cases 0.02 per cent of myopia was found.

The eyes of the child at birth are subjected to considerable risk of injury, especially during instrumental or difficult delivery, and a careful ophthalmic examination should be made of the fundus of each eye. It may be the first evidence of intracranial trauma and the cause of poor vision later in life. Kearney reported that intracranial hemorrhage at birth was responsible for 70 per cent of the spastic type of paralysis in children and 20 per cent of the resulting idiocy and feeblemindedness now existing. These injuries often remain unobserved, and some congenital defects are probably due to this trauma.

The pupillary reaction in cases of intracranial hemorrhage is indefinite and cannot be relied on. It has been said that the dilated pupil, owing to pressure on the third nerve, is to be found on the side of the trauma to the brain. Bunten stated, however, that it is common to find a constricted pupil on the side of the lesion, especially in the early stages, or there may be bilaterally dilated or constricted pupils or no pupillary changes. On the other hand, Cushing stated that ocular palsies and inequality of the pupils at birth is not common. Feinberg claimed that occasionally there may be an inequality of the pupils, the one on the affected side being smaller and the palpebral fissure narrowed. He considered this an important finding and of value in prognosis because it often means injury to the cervical sympathetic nerves. This may be the cause of heterochromia iridis developing later in life. In my series of 458 cases, inequality of the pupils was found to exist 5 times, and all but 1 of these cases showed intra-ocular hemorrhage and evidence of intracranial pressure.

Obstetric histories rarely mention the existence of any ocular injuries unless they are very apparent, and it is quite probable that ocular injuries are more frequent than obstetric data would indicate. Berkeley concluded, however, that ocular disabilities due to injury during the process of birth were rare, and the inquiries of Gifford gave mostly negative results.

Intra-ocular hemorrhages in the new-born absorb quickly without leaving any permanent effects, and all investigators agree that they do not interfere with vision in later life unless a large hemorrhage occurs at the macula, in which structural changes may occur. This may be the cause of amblyopia, as well as congenital nystagmus and degenerative macular changes. Retinal hemorrhages are frequent, especially after difficult delivery, though a large number of apparently healthy children are born with retinal hemorrhages. In this series, retinal hemorrhage was found in 41.7 per cent, and macular hemorrhage was noted 4 times. The more severe macular hemorrhages occurred in cases of difficult labor. The most common distribution of retinal hemorrhage was in the fibrous layer of the retina, usually near the disk and along the vessels. In some cases small hemorrhages extended to the periphery,

but the majority were near the posterior pole. Small, round and darker hemorrhages were noted at times, but the greater number were flame-shaped. Absorption of the hemorrhage usually took place in four or five days. Macular hemorrhages, however, disappeared more slowly, as a rule in about fifteen days. In the rarer cases of macular hemorrhage any changes that may occur may be due to spreading of the retinal fibers or some interference with their development.

Some writers believed that the different types of presentation have a definite relationship to intra-ocular hemorrhage. In the cases examined, with the more common left occipital anterior presentation, hemorrhage occurred in the right eye 28 times, in the left eye 15 times and in each eye 41 times, and it was found more often in the left eye in right occipital anterior presentation.

Eades agreed with Juler that delay in birth, asphyxia, prolonged labor, early rupture of membranes, increased pressure of the head by the forceps or contracted pelves did not increase the tendency to retinal hemorrhage. However, in the cases here presented, retinal hemorrhage was found in the infant in 58 of 95 low forceps deliveries, 49 of these being in primiparas, with bilateral retinal hemorrhage occurring 29 times. High forceps and midforceps deliveries were used 17 times, and severe bilateral retinal hemorrhage was found in every case.

Eades believed that neither anesthesia nor narcosis played a part in retinal hemorrhage, but Jacobs, in his series of cases in which morphine or scopolamine hydrobromide was used, found 63 per cent showing retinal hemorrhage. In this series, gas and oxygen were administered to almost every mother, and solution of pituitary was used in most cases and probably had little if any effect on the incidence of retinal hemorrhage.

Pelvic anomalies and long complicated labors are usually given as a cause of intracranial and intra-ocular hemorrhage, but in this series of examinations no one condition seemed to stand out as being responsible for ocular hemorrhage except in cases of forceps extraction with extra-ocular lesions, and in premature and immature infants. Fifty per cent of the premature babies and 90 per cent of the immature babies showed retinal hemorrhage. Four immature infants delivered by cesarean section, however, did not show any hemorrhage, but three full-term infants delivered by section did show retinal hemorrhage shortly after birth. Rosamond, in 1921, reported a case of intracranial hemorrhage following a similar operation. This is difficult to explain unless some manipulation of the child would cause it.

The few hazy disks seen were no doubt caused by intracranial pressure during the passage of the head through the birth canal and simulated a low grade papilledema. They were seen in the more severe cases of forceps extraction.

A general consideration of retinal hemorrhage in the new-born emphasizes the fact that the condition is often a complex disease syndrome and that it is due not to any single cause but to many causes which may be found in the circumstances governing labor. In most cases this bleeding occurs during the act of birth from the trauma incurred. If microscopic examination could be made of apparently healthy fundi, choroidal engorgement and retinal hemorrhages would no doubt be seen in a large percentage of cases in which no ophthalmoscopic evidence of hemorrhage could be noted. The pressure on the head must cause congestion of the veins, and sudden release after birth probably results in undue dilation of the frail vessels, and bleeding ensues. The blood of the retinal circulation is drained by the central vein, which passes as a single trunk through the lamina cribrosa. Behind the lamina, the vein runs along beside the artery in a thick connective tissue sheath in the center of the nerve. Behind the bulb, it bends out toward the pial sheath and enters the dura obliquely. In the orbit, it joins one of the ophthalmic veins as a rule and enters the cavernous sinus. Any one or all of these veins are apt to be much congested during birth, owing to intense compression, and retinal hemorrhage in most cases depends on the venous congestion.

COMMENT

1. Ocular symptoms are often important in making a diagnosis of intracranial trauma or hemorrhage.

2. Ocular injury at birth may be permanent, or the injury may be only temporary with no destruction of vision.

3. Retinal hemorrhages occur in approximately 41 per cent of cases.

4. The main factor in retinal hemorrhage is probably congestion of the retinal veins due to increased intracranial pressure and a combination of circumstances governing labor.

5. Abnormalities of labor do not increase the tendency to retinal hemorrhage unless high forceps deliveries are used.

6. Direct pressure on the eye by forceps, whether high or low, is always dangerous to vision.

7. Premature and immature infants are apt to show retinal hemorrhage.

8. A careful ophthalmoscopic examination should be made of every infant immediately after birth, and it is especially important after a difficult delivery.

Dr. Howard Johnson, Medical Director of St. Luke's Hospital, assisted in making the necessary arrangements to carry forward my examination of the eyes of the new-born.

SUMMARY OF DATA ON OCULAR CONDITIONS IN THE NEW-BORN

Frequency of Retinal Hemorrhage as Reported by Various Investigators

Author	Date	Mature Infants: Cases Studied	Mature Infants: Percentage of Retinal Hemorrhage	Premature Infants, per Cent
Koenigstein	1884	281	10	..
Schleich	1884	150	32	..
Naumoff	1890	...	25	..
Montalcaine	1897	...	23	..
Paul	1900	200	34	40
DeVries	1901	...	11	..
Sicherer	1909	400	20	32
Stumpf	1905	136	24	32
Coburn	1906	...	46	..
Seefelder	1907	...	45	..
Lequeux	1911	151	25	..
Ehrenfest	1922	...	12	..
Jacobs	1924	100	12	..
Bedell	1924	...	14	..
Juler	1926	...	15	..
Hoffman	1926	...	20	..
Howland	1926	400	3	..
Lachman	1927	73	17	..
Seissiger	1929	500	19	..
Cortelini	1929	150	23	..
Eades	1929	138	17	..
Sykes	1931	...	23	..
Edgerton	1933	458	41	70

Bilateral Retinal Hemorrhage as Reported by Various Investigators

Schleich	1884	150	59
Sicherer	1909	400	35
Seissiger	1929	500	10
Edgerton	1933	458	12.6

*Number of Infants Examined**

First Day	Second Day	Third Day
434	23	1

Ocular Conditions Found at Birth

Extra-ocular lesions	23
Retinal hemorrhage	191
Pupillary membranes	27
Hazy disks	6
Squint	8
Number of forceps extractions	95

Gestation Time in Weeks

Average	Minimum	Maximum
36½	28	40

Presentation in Relation to Hemorrhage

	Primipara	Multipara	Right Eye	Left Eye	Both Eyes
L. O. A.	57	27	28	15	41
L. O. P.	6	3	3	3	3
R. O. A.	12	8	7	9	4
R. O. P.	5	..	1	1	3
Breech	4	4	2	2	4
Brow	3	..	..	..	3
Forceps	49	9	14	15	29
Cesarean section	2	1	2	1	

Retinal Hemorrhage in Relation to Parity and Labor

Infants with retinal hemorrhage whose mothers were under 25 years	51
Infants with retinal hemorrhage whose mothers were over 25 years	90
Primiparas whose infants showed retinal hemorrhage	138
Multiparas whose infants showed retinal hemorrhage	52
Primiparas whose infants showed no hemorrhage	177
Multiparas whose infants showed no hemorrhage	144
Number of first pregnancies	264
More than one pregnancy	191
Delivery in first 12 hours	155
Delivery in 24 hours	222
Delivery after longer period of labor	78
Contracted pelves	40
Ages	from 17 to 42
Length of labor	from 1 to 72 hours
Weight of mothers:	
Average	134 pounds
Minimum	93 pounds
Maximum	219 pounds

SUMMARY OF DATA ON OCULAR CONDITIONS IN THE NEW-BORN—*Continued*

Miscellaneous Data

Ocular examination of the mothers: 10 myopic; remainder either emmetropic or hyperopic

Retinal hemorrhage in cases of asphyxia.....All but 2

Wassermann test in all cases of retinal hemorrhage.....Negative

Sex and Maturity

Males	Females	Twins	Full Term	Premature
238	220	3	438	20

Weight of Mother and Infant

	Average	Minimum	Maximum
Baby's weight:			
Boys.....	7.6	4.6	11.10
Girls.....	7.3	3.9	10.14
Mother's weight.....	134	93	219

Mother's Age

Average	Minimum	Maximum	Under 20	From 20-30	From 30-40	Over 40
26.6	17	42	27	312	113	2

Hours of Labor

	Average	Minimum	Maximum	Under 10	From 10-15	From 15-20	Over 20
Primiparas.....	23.3	10	72	..	5	31	162
Multiparas.....	10.8	1	27	50	69	27	12

Type of Presentation

Cephalic	Breech	Face or Brow
436	19	3

Type of Delivery

Normal	Forceps	Breech	Section
308	97	19	34

Position

L. O. A.	L. O. P.	R. O. A.	R .O. P.	Breech	Brow
298	15	65	24	19	3

Condition of Pelves

Normal	Contracted	Flat
417	31	10

* The data from this point on through the table refer to the author's series of 450 newborn infants.

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Obituary

L. MAUD CARVILL, M.D.

In the death of L. Maud Carvill the community and the medical profession have suffered a severe loss, not wholly from a professional standpoint, but because there has left our midst one of the finest characters that New England has produced. Dr. Carvill was dignified when dignity was necessary and joyous when joyousness was best. She was always cheerful, with a ready smile for all those with whom she came in contact. "Ever calm and always gentle, she moved about her world, serene, cheerful, patient. One of God's gentlewomen, fine and lovely in character, strong and faithful as a friend." She always gave of her best to her host of loyal patients and friends.

She was graduated from Tufts College in 1899 and from Tufts College Medical School in 1905. She brought to the specialty of ophthalmology a quiet determination and effectiveness that were extremely fruitful. For many years she was assistant surgeon at the Massachusetts Eye and Ear Infirmary. She was also a member of the ophthalmic staff at the New England Hospital for Women and Children and was later consultant in ophthalmology for that institution. For several years she was an instructor in ophthalmology at Tufts College Medical School.

She was a member of the following societies: the American Medical Association, the New England Women's Medical Society, the American College of Surgeons, the American Academy of Ophthalmology and Otolaryngology, the New England Ophthalmological Society and the American Ophthalmological Society. She had the unique distinction of being the only woman ever to be admitted to membership in the American Ophthalmological Society.

It can be truly said that she was for many years, as shown by her writings and her society memberships, the leading woman ophthalmologist of America. Her research work was internationally known and its worth recognized. Her research work at the Massachusetts Eye and Ear Infirmary was of outstanding character, and she will long be remembered for this, especially by those who worked with her. The late Dr. George S. Derby placed great reliance on the painstaking character of her services and on her honest deductions.

The following is the list of her publications:

Congenital Fistulae of the Lachrimal Canaliculi, Arch. Ophth. **38**:585, 1909.
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News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Advisory Board for Medical Specialties.—In order to elevate the standards of ophthalmology, to improve the efficiency of those claiming in their practice to be ophthalmologists and to standardize the training of eye specialists, the American Board for Ophthalmic Examinations was formed in 1915. It was the outgrowth of considerable study of committees named by the American Ophthalmological Society, the Section of Ophthalmology of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology. This board is the pioneer among the special boards. It consists of nine members, three from each of the component societies. Its members are chosen in the same manner as the presiding officers of the national societies, and members can serve for no more than six years continuously. The board's name was changed in 1933 to the American Board of Ophthalmology to conform with the names of the other boards existing at that time. The other specialties which have already established boards are otolaryngology, obstetrics and gynecology, dermatology and pediatrics. There is now under consideration the formation of boards in radiology, orthopedics, anesthesia, urology, etc.

In 1932 there was organized an Advisory Board for Medical Specialties whose purpose is to coordinate and cooperate with the various official groups interested in postgraduate education of specialists and to bring about a standardization of methods and requirements for the certification of specialists. This board is composed of representatives from the following organizations: the American Hospital Association, the Association of American Medical Colleges, the Federation of State Medical Boards of the United States, the National Board of Medical Examiners, the American Board of Ophthalmology, the American Board of Otolaryngology, the American Board of Obstetrics and Gynecology, the American Board of Dermatology and Syphilology and the American Board of Pediatrics. Examining boards in other specialties will be eligible to representation when they meet certain requirements.

The officers, elected in February, 1934, at Chicago are as follows: president, Dr. Louis B. Wilson, Rochester, Minn.; vice president, Dr. J. S. Rodman, Philadelphia; secretary and treasurer, Dr. Paul Titus, Pittsburgh; executive committee, Dr. William P. Wherry, Omaha, and Dr. Walter B. Lancaster, Boston.

One of the aims of the Advisory Board for Medical Specialties is to act in an advisory capacity in problems of undergraduate education as well as of graduate instruction in the various specialties represented; also it will assist the Council on Medical Education and Hospitals in investigating and listing postgraduate facilities in America and to some degree abroad; it should thus be an important influence in effecting general elevation of the standards of training and practice of the various specialties.

It has been seriously suggested that the time will come when the various states will license physicians to practice as specialists and that the certificates of the various boards will be the basis of such licensure. One Canadian province has already instituted licensure of all its specialists.

It is intended that the Advisory Board for Medical Specialties will cooperate with the Council on Medical Education and Hospitals of the American Medical Association. The next edition of the American Medical Directory is designed to publish information about all accepted special boards and will list conspicuously the names of those specialists who have been found to be competent and who have received the certificate of such special boards.

There are plans under advisement for a directory of all certificated persons; this directory will also list postgraduate training facilities, residencies, internships, etc., in the various specialties as well as the general qualifications for certification.

A meeting of the Advisory Board of Medical Specialties will be held at Cleveland on Sunday, June 10, 1934, immediately prior to the annual meeting of the American Medical Association.

WILLIAM H. WILDER, M.D., *Secretary*,
American Board of Ophthalmology.

Work of National Society for Prevention of Blindness.—The recently published annual report of the National Society for the Prevention of Blindness discloses the progress made by this society in its work of controlling the chief causes of blindness and reducing ocular accidents to a minimum. The work of the society has been carried into the medical and nursing professions, social work, education, industry, organized labor and federal, state and local governmental departments. During the past year, a staff member was engaged to work with teacher training institutions, in an effort to guide classroom teachers toward participation in the school program of conservation of sight. Thirteen young women have been trained in the medico-social aspects of this work. Demonstration centers were conducted during 1933 at the Wills Hospital in Philadelphia, the Charity Hospital in New Orleans and the Washington University Clinics and Allied Hospitals in St. Louis. At present the society is cooperating with the American Medical Association, the American Social Hygiene Association and other organizations in a campaign against syphilis as a cause of blindness. In connection with this movement, it is worth noting that ophthalmia neonatorum, commonly known as babies' sore eyes, has been reduced 75 per cent in the last twenty-five years. Five states have made it mandatory for automobiles to be equipped with nonshatterable glass, and the use of this glass in the spectacles of young people is rapidly becoming general. The growth of the sight-saving movement during the past few years may be readily appreciated when one notes that the original few "sight-saving" classes in the United States have now increased to 430.

SOCIETY NEWS

The Annual Congress of the Ophthalmological Society of the United Kingdom was held at the Royal Society of Medicine, London, April 12 to 14, 1934. The president of the society, Mr. M. S. Mayou, gave the opening address, which was devoted to: "The Surgical Treatment of the More Unusual Forms of Squint and Heterophoria with Especial Reference to End-Results." Dr. F. Bernard Chavasse opened the discussion.

PERSONAL

Associates of Dr. Harry Friedenwald, emeritus professor of ophthalmology of the University of Maryland School of Medicine, Baltimore, gave a surprise dinner in his honor on February 3. About forty physicians were present, including members of the staff of the Baltimore Eye, Ear and Throat Hospital, with which Dr. Friedenwald has been associated for thirty-six years.

UNIVERSITY NOTES

The Smith-Reed-Russell lecture for March at the School of Medicine, George Washington University, was given by Dr. John Wheeler, professor of ophthalmology at Columbia University and director of the Ophthalmological Institute. Dr. Wheeler spoke on "Exophthalmos."

On July 1, Dr. Alan C. Woods will succeed Dr. William H. Wilmer at the Johns Hopkins University School of Medicine and the Wilmer Ophthalmological Institute. Dr. Woods will then become professor of ophthalmology at the university and acting director at the institute.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

LACRIMAL MUSCLE OF DUVERNEY-HORNER. R. GALLENGA, Rassegna ital. d'ottal. **1:** 96 (Jan.-Feb.) 1932.

The author once again, after careful anatomic researches, affirms the independence of the lacrimal muscle of Duverney-Horner from the palpebral orbicularis muscle.

Embryologically or genetically they have no relation.

The time of its first appearance, its growth and differentiation, its origin from a point where undoubtedly myoblasts of the internal rectus muscle exist and its subsequent relations with the anterior expansion of Tenon's capsule represent a complex of data which should have been accurately evaluated by those who uphold its derivation from the orbicularis. More probably it is genetically allied to the internal rectus muscle.

Even macroscopically the lacrimal and the orbicularis muscles differ in morphology and topography.

From morphologic data it must be admitted that the lacrimal muscle acts not as much on the lacrimal sac as on the canaliculi, and particularly on the inferior canaliculus and caruncle. The lacrimal muscle has no connections with the straight tendon of the orbicularis, but only, and in a limited way, with its reflex tendon.

Gallenga has never seen an anterior lacrimal muscle as a distinct anatomic entity. The muscle is probably a bundle of fibers from the orbicularis muscle, which unites anteriorly with the lacrimal sac and with the straight tendon of the orbicularis itself.

A series of excellent plates illustrates the essay.

V. R. SYRACUSE.

Bacteriology and Serology

INJECTION OF AUTOGENOUS BLOOD INTO THE ANTERIOR CHAMBER OF EYES WITH TUBERCULOSIS OF THE IRIS. ILSE BAER, Klin. Monatsbl. f. Augenh. **90:** 485 (April) 1933.

The author renders a detailed account of the research, theory and therapy of injections of the patient's own blood into the anterior chamber in tuberculosis of the iris after the method of Schieck. Experience with implantation of portions of iris, obtained by an iridectomy, in the anterior chamber of the rabbit's eye is recorded. It was found that no reaction followed implantation of portions of tissue from chronic recurring iritis, but that tuberculous manifestations developed when the tissue was taken from severe, mostly unilateral, conglobating tuberculosis of the iris. These experiments failed to produce manifestations corresponding to the severity of tuberculosis of the iris in man. The author then reports the result of her experiments at Schieck's eye clinic at Würzburg. She injected 0.1 cc. of a solution containing virulent

tubercle bacilli into the anterior chamber of four rabbits. Two of them showed tuberculous changes after eighteen days; two, after one month. The first group received injections of 0.1 cc. of their own blood into the anterior chamber; the second group were given no injections of blood. The symptoms were the same in each instance, and the course of the disease was not influenced by the injections. The therapeutic failure was due, in the author's opinion, to the absence of antibodies in these highly virulent primary infections.

The presence of antibodies, on the other hand, and perhaps also the action of irritant bodies in the blood are responsible for the favorable results obtained with injections of autogenous blood in all forms of chronic tuberculosis of the iris. The results were limited by complications of the posterior uvea, especially perforating, conglobulating tuberculosis of the uvea or tuberculous panophthalmia.

K. L. STOLL.

Color Sense

RESEARCH ON THE RAYLEIGH EQUATION ON NAGEL'S ANOMALOSCOPE IN THE DETERMINATION OF RELATIVE DALTONISM. G. COLAJANNI, Ann. di ottal. e clin. ocul. **61:** 99 (Feb.) 1933.

The anomaloscope, considered the most accurate practical means of determining color vision, was employed in the examination of seventy-nine air pilots or candidates. Of sixty-seven who had passed normal tests with Holmgren's wools and the pseudo-isochromatic plates of Ishihara, fifty-four showed normal equations with the anomaloscope, while thirteen showed variations beyond the normal limits of error and could hence be considered anomalous trichromats. Those with vision defective for green (deuteranomalous) were much more numerous than those with vision defective for red (protanomalous). (The figures are confusing.) Of twelve subjects who showed red-green blindness with the wools and Ishihara plates, six showed equations with the anomaloscope which could be considered normal.

The investigator believes that tests with the anomaloscope should be controlled by other tests corresponding more nearly to the practical use of color vision. In tests for the air service the anomaloscope is employed, but when anomalies are found, the subjects are submitted to further examination with a special chromoptometer which measures the perception of colored lights of low intensity seen under the minimum visual angle and for periods as short as one sixtieth of a second; such tests are considered of the greatest importance in determining a candidate's fitness for the service.

S. R. GIFFORD.

Conjunctiva

OCULAR PEMPHIGUS. W. M. JAMES, Am. J. Ophth. **15:** 815 (Sept.) 1932.

Two cases of ocular pemphigus are reported, the first occurring in a white man, aged 57, and the second in a Negress, aged 66.

In both cases, aside from the usual clinical manifestations, the biomicroscope showed that in the areas in which the conjunctiva seemed to be normal the superficial capillaries were surrounded by a relatively thick sheath of white perivascular tissue which was seen to exert traction

whenever the eyelids moved. In the vicinity of the grossly visible scar tissue there was a substantial increase in the perivascular tissue. The capillaries, arterioles and venules showed an irregularity much greater than is normally seen in the conjunctival circulation.

The author gives the following summary:

Ocular pemphigus may occur in association with similar lesions of the skin or other mucous membranes than that of the eye. It may also occur primarily as an ocular disease.

The phytopharmacologic test of Pels and Macht is an aid in the diagnosis of the disease, in the early stages particularly, and indicates that the disease is probably the result of a toxin circulating in the blood stream.

Biomicroscopic examination of the lesions of pemphigus indicates that perivascular fibrosis occurs before the formation of vesicles.

The treatment of pemphigus is of little avail. A bibliography is appended.

W. ZENTMAYER.

ULCEROVEGETATIVE CONJUNCTIVITIS WITH CONSIDERABLE GLANDULAR DISEASE; METHYLIC ANTIGEN; CURE. F. ROUSSEAU and J. SPILLMANN, Ann. d'ocul. **170:** 333 (April) 1933.

The attention of the authors has been drawn to recent articles on conjunctival enlargement with a ganglionic reaction, too often designated by the general term of Parinaud's conjunctivitis, which should not be used except for a primary tuberculous lesion of the conjunctiva, sporotrichosis or granuloma. This term granuloma comprises different conjunctival infections with an ulcerovegetative reaction, some of which are unknown. In the case reported the presumption of a tuberculous etiology is strong; however, the absence of inoculation tests prevents the authors from confirming this etiology. The case and the treatment used are reported in detail.

S. H. MCKEE.

Comparative Ophthalmology

NOTES ON THE LATERAL EYES OF SPHENODON, WITH SPECIAL REFERENCE TO THE MACULAR REGION. I. MANN, Brit. J. Ophth. **17:** 1 (Jan.) 1933.

Sphenodon (one of the order Rhynchocephalia) occupies an intermediate position between the tortoises and the crocodiles.

A short account of the animal is given, followed by a description of the external and internal structures, including the ophthalmoscopic and biomicroscopic appearances and the histology of the eye.

With regard to the retina, it can be said that over the greater part of its extent its stage of differentiation corresponds to that seen in a 110 mm. human fetus. In the early stages the macula resembles the developing human macula in a striking manner.

The author makes the following interesting comments: It may appear remarkable that two species so widely separated as the primates and Rhynchocephalia should show such strong resemblance in both embryonic and adult structure in what might have been considered as one of the most highly specialized regions of the body. The fact may possibly be interpreted as showing how early a retinal area specialized

for form vision appears, or that the human macula may be added to the lengthening list of man's primitive characters. It is interesting to note how, on the one hand, one finds in birds extreme elaboration of the macula and extension and even multiplication of the site, while, on the other hand, in most mammals (excluding the primates) there is either absence of the fovea altogether or extreme simplification. It is as if the primitive potentialities for accurate form vision fell into abeyance in the lower mammals only to be reestablished again with the acquisition of binocular vision. That the position of the eyes had originally nothing to do with the specialization of a fovea is obvious from its presence in Sphenodon, which cannot have any binocular vision, in birds in which both binocular and uniocular vision is found, and in the primates with true binocular and stereoscopic vision. The abeyance of the fovea in the lower mammals is probably, therefore, a correlate not of the position of their eyes but of the subsidiary part played by vision, which in them is overshadowed by the rise in importance of the sense of smell. Thus the microsmatic visual human type comes to resemble the primitive ancestor more closely than its own nearer relations.

W. ZENTMAYER.

Cornea and Sclera

EPIDEMIC SUPERFICIAL KERATITIS. S. SANYAL, Am. J. Ophth. **16:** 390 (May) 1933.

The author discusses acute superficial keratitis and gives the data of an epidemic in Calcutta during the monsoon period in the summer of 1932. He describes the different types encountered and gives the following conclusions:

"It will be observed that this epidemic is different from that at Madras in course, prognosis, and complications. It is true that no careful study of the etiology of the epidemic has been made, but even if the two epidemics—the one at Calcutta and the other at Madras—prove to be due to the same causative agent, the clinical description of a first epidemic under different climatic conditions might prove to be useful."

W. S. REESE.

DERANGEMENT OF THE CORNEAL NERVES. J. H. DOGGART, Brit. J. Ophth. **17:** 385 (Aug.) 1933.

According to Doggart, there is no need to assume the presence of special trophic elements in the nerves. All nerve fibers may be regarded as trophic. The epithelial cells of the cornea are extremely sensitive toward any interference with their nerve supply, but most of the clinical evidence suggests that abnormal impulses derived from unhealthy nerve cells or fibers are chiefly responsible. Mere severance of normal nerve connections does not, as a rule, provoke serious corneal lesions, although it can predispose to such lesions by abolishing protective reactions.

The nerve communications of the cornea become disordered not only in conditions that are primarily neural, but also in any form of acute corneal infiltration. In most cases of hypopyon ulcer, the whole surface of the cornea is insensitive to touch. The same is true in cases of acute glaucoma.

In herpes zoster ophthalmicus, the association of ocular lesions with damage to the gasserian ganglion is established. In neuroparalytic ulceration of the cornea, a peripheral as well as a central factor must be present. In herpes febrilis, it is probable that the nerves involved are the interepithelial plexuses in localized areas. There is a fairly close affinity between the behavior of herpes febrilis and that of recurrent abrasions. Trauma may be considered as a precipitating factor, not the essential cause. In the miscellaneous collection of diseases grouped under the name of superficial punctate keratitis, it is highly probable that some lesion of the nerves is frequently responsible. The corneal lesions met with during convalescence from influenza are probably due to the toxic effect of the influenza organisms on the corneal nerve supply.

The corneal dystrophies, including nodular keratitis, are possibly due primarily to diseases of the nerves. Other conditions of possible origin in the nerves are bullous and band keratitis and marginal degeneration of the cornea.

Most of the disorders in which for no evident reason the nutrition of the cornea declines are associated with gradual diminution of the sensibility of the cornea. To this rule keratoconus is an exception, for it is commonly accompanied by hyperesthesia, which suggests that the increased visibility of the corneal nerves in this condition is an expression of their actual hypertrophy.

It is suggested that as epithelial edema is the most characteristic early sign of disordered corneal nerve supply, it is necessary to consider the possibility of a lesion of the nerves in persons who persistently show this sign without any rise of ocular tension and in the absence of any obvious traumatic or inflammatory cause. W. ZENTMAYER.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF MEGALOCORNEA, AND ITS PIGMENT DISPERSION, INCLUDING THE KRUNKENBERG-AXENFELD SPINDLE. B. KAYSER, Klin. Monatsbl. f. Augenh. **89:** 770 (Dec.) 1932.

The question of whether megalocornea occurs as an idiogenous, congenital anomaly has been settled in the affirmative by the publication of two family trees, one reported by Kayser with bilateral megalocornea in seventeen members, and the other by Groenholm with the same condition occurring in thirteen members. Complete coincidence of the symptoms was found in these sixty eyes. Hydrophthalmos was absent. Only males were afflicted, and every case was bilateral and complicated by regular astigmatism. The radius of the curvature of the cornea was equal to or smaller than the normal average, and the anterior chamber was deep. All the eyes were free from pathologic changes and the subjects were healthy. Kayser, in commenting on the typical appearance of eyes with megalocornea, quoted a similar impression gained by Groenholm, who remarked on their brightness and beauty in contrast with hydrophthalmic eyes.

The symptoms enumerated, observed also by Gerty in the case of two brothers who, like two brothers of their mother, had megalocornea, are sufficient in totally excluding hydrophthalmos; their presence or absence are valuable aids in establishing a differential diagnosis. In a

single instance the malformation was transmitted from mother to daughter in an otherwise gynophore type of heredity.

In one of Kayser's patients gerontoxon began at the age of 10, and bilateral Krunkenberg-Axenfeld spindle at the age of 23 in one eye, at the age of 25 in the other eye. These two eyes, furthermore, showed irregularly scattered pigment on the anterior surface of the iris, especially in the furrows which form on contraction of the iris. This phenomenon, never described before, and his observation that the spindle increased in width when a large amount of pigment was present prompted Kayser to the opinion that the spindle is no firmly established formation. He thinks that the pigment originated from the posterior surface of the iris, settled loosely on the posterior surface of the cornea, and was carried by the circulation of the aqueous humor to the anterior surface of the iris. Particles of pigment of the spindle embedded in the endothelial cells of the cornea, of course, will remain there. The reason cannot be explained why this scattering should take place only in the otherwise healthy eyes with megalocornea, but not in hydrophthalmos.

K. L. STOLL.

ANATOMIC MEASUREMENT OF EYES WITH MEGALOCORNEA. B. KAYSER, Klin. Monatsbl. f. Augenh. **90:** 626 (May) **91:** 343 (Sept.) 1933.

In the first of the present articles, Kayser refers to his previous article (*Klin. Monatsbl. f. Augenh.* **89:** 770 [Dec.] 1932) in which he discussed the difference between congenital hydrophthalmos and megalocornea. In the present publication he sought to establish whether, in megalophthalmos or in gigantophthalmos, the globe is necessarily enlarged in proportion to the measurements of the megalocornea. Cases of Hartleib, Berg, Ferber and others are cited to show that the theory that a proportionate enlargement occurs is erroneous. Hartleib's case, which seemed to support the theory, is considered atypical, and the eyes which Berg described as "macrophthalmi" belong, according to Kayser, in the realm of the variability of the eye of normal size. The type of megalocornea which protrudes from the eye "like a dome" and which is inherited in a strictly gynophor manner should be termed "megalocornea globosa," as suggested by Horner and Kayser and recently by Berg. In this article, Kayser came to the following conclusion: "It seems certain that usually a globe of normal size is found in megalocornea, gigantophthalmos does not occur, macrophthalmos is doubtful." After publication of this article he examined the typical megalocornea of a relative of the patient on the observation of whom for twenty-two years he based the foregoing statement. Although the globe appeared to be of normal size at first glance, the perpendicular diameter measured 24 mm., the horizontal, 24.5 mm., and the sagittal, 27 mm., taken anteroposteriorly. Hence, the cornea was not located within the circle of the globe, but protruded considerably beyond the periphery. The diameter of the cornea was 14 by 14.5 mm. and the depth of the anterior chamber was 7.5 mm. These measurements corroborated Kayser's statement that the enlarged cornea rests as a calotte on a globe of normal size. His conclusions, published in the second article, are: An eye with megalocornea is a disproportionate eye and different from proportionately

enlarged eyes, such as the "macrophthalmi" which Berg described; these two types should be distinctly separated. The unequal development of the cornea and the remainder of the globe may be explained by Waardenberg's contention that a number of hereditary units govern the development of the eye.

K. L. STOLL.

Experimental Pathology

PATHOGENESIS OF "RHINOGENOUS" NEURITIS: AN EXPERIMENTAL STUDY. M. A. WOLFKOVITCH, Sovet. vestnik oftal. **2**: 405, 1933.

The investigator doubts the rhinogenous origin of numerous cases of optic neuritis reported as having this origin. The enlargement of the blind spot, the contraction of the visual fields and the central scotomas are common in various forms of optic neuritis; i. e., these changes are not pathognomonic of rhinogenous neuritis. In cases in which the nasal and paranasal cavities are clinically normal the success of endonasal treatment may be explained by its effect on the reflectory system of the nasal mucous membrane. It possibly acts indirectly on the disturbed vasomotor system of the optic nerve.

In order to ascertain the possibility of producing inflammation of the optic nerve by endonasal interference, Wolfkovitch made two series of experiments. The only similar experimental studies, three in number, made in Germany and Japan, had given negative results.

The first series of experiments was carried out on ten dogs, the nasal cavities of which were tamponed. The animals were given daily intravenous injections of a 5 per cent solution of carmine, on the basis that this colloidal dye settles easily on inflamed tissue. One gram of the dye per kilogram of body weight was injected into both experimental and control animals. The dogs were killed within from seven to twelve days. All showed a purulent inflammation of the lacrimal sac and of all the sinuses. There was necrosis of the mucous membrane and of the superficial layers of the nasal bone and of the nasopharynx. There was intensive coloring of the mucous membrane of the nasal cavities of the bone with the carmine. However, no coloring of the optic nerve or of its sheaths was observed.

In the second series of experiments the inflammatory condition of the nasal mucous membrane was produced by filling the nasal cavity with powdered blister beetles (Spanish fly). In addition, the nasal cavity was swabbed with cantharidin. Intravenous injections of carmine were made in the same manner as in the first series. The animals, killed on various days, showed marked inflammation of the nasal cavities and of the sinuses, particularly of the frontal sinuses. The animals killed on the twelfth day showed the same necrotic changes as the animals of the first series. The mucous membrane of the nose and sinuses was colored intensively by the dye. There was also an accumulation of carmine in Havers' canals. The spinal fluid was pinkish. The structure of the optic nerve remained unchanged and contained no granules of carmine.

Thus, this attempt at producing "rhinogenous" neuritis experimentally was also unsuccessful.

O. SITCHEVSKA.

General

THE TRANSMISSION PROPERTIES OF TINTED LENSES. W. W. COBLENTZ, Am. J. Ophth. **15:** 932 (Oct.) 1932.

The author shows the necessity for standardization of tints. Only the dark shades of glasses that absorb the violet end of the spectrum increase the visibility of details of objects which are obscured by the intervening blue haze. For chronic sufferers from glare the light shade that is satisfactory for a glare of threshold intensity will not be dark enough for intense glare. On the other hand, the average person needs dark glasses only for intense glare.

The claim that tinted glasses do not distort colors is substantially correct as regards the light shades as the glasses are practically colorless. Dark shades necessarily alter the color values.

From an examination of the color vision of two patients who had worn tinted lenses for a number of years it does not appear that atrophy of the retina as a result of the deprivation of violet and ultraviolet rays is so serious as the advertisements make it appear.

The author suggests that the number of shades be reduced to three (designated A, B and C). The spectral transmission of seven types of tinted lenses is given.

The practical conclusions drawn are that only the darker shades of tinted lenses can shield the eye from intense glare of visible radiation, and that the protection of the eyes from solar ultraviolet radiation reflected from ordinary surroundings is of secondary importance. In the presence of artificial sources of ultraviolet radiation, used for healing purposes, dark glasses which are opaque to ultraviolet rays should be worn to avoid injury.

W. ZENTMAYER.

General Diseases

VISUAL FIELDS, BLIND SPOTS, AND THE OPTIC DISKS IN ENDOCRINE DISEASE. L. L. MAYER and H. R. RONEY, Am. J. Ophth. **15:** 1024 (Nov.) 1932.

This is an ocular study of fifty patients with endocrine disturbance, seen over a period of two years. There were no abnormal findings, even in cases showing the most evident and serious endocrine disease. The author's conclusions are: There were no characteristic changes in the visual fields for white, in the blind spots or in the color or nature of the optic disks in this series of endocrine disorders.

W. ZENTMAYER.

OCULAR COMPLICATIONS IN A CASE OF AGRANULOCYTIC ANGINA. H. BARKAN, Am. J. Ophth. **16:** 406 (May) 1933.

The author gives a short history and description of agranulocytic angina, and cites the case of a woman, aged 73, whose eyes had been sore for ten days preceding admission. She also had an acute nasal discharge, sore throat, cough and fever. Examination revealed acute pharyngitis and tonsillitis; there were some diseased, dirty teeth; the lips were dry, and the surface of the tongue was caked with yellow patches of mucus. The uvula showed a yellow ulcerated patch with a surrounding diffuse stomatitis. The blood count showed only 1,000

leukocytes per cubic millimeter and no polymorphonuclears. The urine was heavily loaded with albumin and sugar. The patient became worse, but following treatment with pentose nucleotides showed decided improvement; the polymorphonuclear count rose to 57 per cent and the leukocytes to 6,100. Throughout this period the eyes became worse. The original edema of the lids and conjunctival discharge increased, and ten days after admission each cornea showed a beginning ring abscess, which progressed until both corneas were necrotic. The patient recovered but was blind in both eyes.

W. S. REESE.

SHELL-SHAPED PROLIFERATIONS OF THE ENTIRE UVEAL TRACT WITH INVOLVEMENT OF THE ORBIT: A CLINICAL CONTRIBUTION TO ALEUKEMIC LYMPHADENOSIS AND LYMPHOSARCOMATOSIS OF THE EYE; REPORT OF TWO CASES. G. B. BIETTI, Klin. Monatsbl. f. Augenh. **90:** 308 (March) 1933.

A child, aged 3 months, showed extensively swollen glands of the left side of the neck and occiput and enlargement of the liver and spleen. A whitish-yellow mass, adjacent to Descemet's membrane, covered nearly the whole iris of the left eye. The intra-ocular tension was increased. Histologic examination revealed infiltration of the choroid with round cells, larger than lymphocytes and resembling lymphoblasts. The mass in the anterior chamber was an exudate consisting cheifly of decayed infiltration cells. Similarly infiltrated zones were found around the posterior ciliary nerves and vessels, extending into the orbit, and near the sclera in the orbital tissue.

The second case, that of a man, aged 48, presented extensive edema of the lids of the left eye, which extended almost to the ear. This swelling developed within a year after the patient had discovered a small red spot in the conjunctiva. The orbit was exenterated, and masses extending into the nasal cavities were found, which had eroded the bone. Several types of cells, especially lymphoblasts, dwarf cells and large cells with a very large nucleus, were found histologically. No metastases were noted at the time of the operation, but resection of the angle of the jaw became necessary, and death from involvement of the liver occurred four months later. A combination of undoubted symptoms of sarcoma and aleukemic lymphoma prompted the author to consider this case as a combination of these two diseases.

K. L. STOLL.

Glaucoma

ALKALESCENCE OF THE BLOOD IN GLAUCOMA. A. BIFFIS, Ann. di ottal. e clin. ocul. **61:** 109 (Feb.) 1933.

In a work previously reviewed Biffis reported a slight increase in the potassium/calcium quotient of the blood in glaucoma and concluded that the diminution of calcium and increase of potassium favor dissociation of the hydroxyl ions, resulting in relative alkalescence of the tissue fluids. He now reports estimations of the carbon dioxide-combining power of the blood by the Van Slyke method in twenty normal persons between 52 and 82 years of age and in fifty glaucomatous patients of a similar range of age, except for two patients with buphthalmos and juvenile glaucoma. The normal group showed an alkaline reserve

varying between 70.45 and 51.33 and averaging 60.21, while in the group with glaucoma this reserve varied between 77.7 and 50.52, averaging 62.31. Biffis thinks that the values in glaucoma, while within the limits of normal, show a slight displacement toward the alkaline side. He offers certain theoretical explanations of the dilatation of the pupil and the rare occurrence of glaucoma in myopia based on his findings. (It seems doubtful to the reviewer that the conclusions are justified by such slight deviations from the normal.) S. R. GIFFORD.

END-RESULTS AFTER CYCLODIALYSIS. E. BUNGE, Klin. Monatsbl. f. Augenh. **90**: 21 (Jan.) 1933.

Bunge reports the results obtained with cyclodialysis on 320 eyes operated on at the University Eye Clinic at Kiel, most of them by Heine. Two tables render a classification of the types of chronic glaucoma and of the result. The first table, comprising the entire material, shows that in about one fourth of the cases cyclodialysis was combined with, or had to be followed by, iridectomy, anterior or posterior sclerotomy, puncture of the anterior chamber or repetition of cylodialysis.

The second table refers to 166 of these eyes, which were observed for periods of from one to four and more years. Tension was improved and vision remained stationary in 68.9 per cent of simple and of chronic inflammatory glaucoma; cyclodialysis had to be repeated or was combined with other operations in 23 per cent of the first named type, and in 40 per cent of the second. Cyclodialysis was considered indicated whenever vision decreased and the field of vision contracted under treatment with miotics, while less importance was attributed to the intra-ocular tension. It is uncertain whether a communication between the anterior chamber and the suprachoroidal lymph space resulted in every case in which the operation was successful, although Elschnig found it present in an eye which was examined histologically fourteen years later. The communication was absent in experiments on animals and in human eyes which were removed after unfavorable operations.

Two more tables give a synopsis of the results of other operators, the second of these showing a comparison of the number of cyclodialysis and trephining operations performed by the same five authors between 1918 and 1931. All of the authors but one favored cyclodialysis more from year to year.

Bunge considers cyclodialysis equal to the trephining operations as far as the results are concerned, and superior to them in preventing later complications. Cyclodialysis should be the operation of choice in chronic glaucomas; trephining operations should be performed after cyclodialysis has failed. K. L. STOLL.

Hygiene, Sociology, Education and History

THE EYES OF SCHOOL CHILDREN. E. JACKSON, Am. J. Ophth. **15**: 901 (Oct.) 1932.

The author outlines the procedure for a complete examination of the eyes of school children. He describes the method of determining the visual acuity demonstrated by Brown and Royer and emphasizes its

practicability. Next in importance is the ophthalmoscopic examination. Skiascopy, without cycloplegia, rapidly gives information of real value, revealing any gross heterophoria or weakness of ocular movement that calls for attention. If there is any reason to suspect that the field of vision may be defective the best test is the confrontation test, in which the child is required to tell whether the fingers are held still or are moving in the periphery of the field. W. ZENTMAYER.

THE DEVELOPMENT OF GLASSES. N. BLATT, Miscarea med. **5:** 601, 1932.

Roger Bacon is said to have contrived spectacles in the thirteenth century, but probably the Romans knew the use of lenses earlier. This is pointed out in the second century A.D. in the older Roman works of Seneca, Cicero, Cornelius Nepos and Cornelius Ptolemaeus, to whom refraction was already known. The Egyptian physician Alhazan was aware of the magnifying power of convex surfaces.

It is open to question whether the Chinese of that epoch knew anything of the use of spectacles. The statues of stone or bronze found in the ancient Buddhist temples could give one light on this matter. On the photograph of a statue of Buddha the author was able to recognize a spectacles-like object in front of the eyes.

The first form of spectacles, known from a picture, was that of the so-called clinched spectacles. The further evolution of spectacles in chronological order was as follows: the forcipate spectacles in the fourteenth century, the arciform spectacles in the fifteenth century, the "one-glass" in the sixteenth century, and the forehead-bar spectacles, the small spectacles of the Biedermeier period (from about 1816 to 1848). Not until the second half of the nineteenth century did the temple spectacles make their appearance. This type, except for a few modifications, is retained up to this time.

Some years later appeared the contact glasses which are applied directly to the globe and are the starting point in a further evolution of spectacles. N. BLATT.

Injuries

CONTUSION OF THE EYEBALL WITH DELAYED INTRAOCULAR HEMORRHAGE. F. C. CORDES and W. D. HORNER, Am. J. Ophth. **15:** 942 (Oct.) 1932.

Three cases are reported illustrating the fact that contusions of the eye complicated by hyphemia must be regarded as potentially serious. All three cases appeared to be instances of simple contusion with hyphemia. However, within from three to five days after the injury, marked recurrent hemorrhages occurred even though absorption of the primary hyphemia was taking place. The source of the secondary hemorrhages is speculative. The primary bleeding probably came from the iris. A secondary hemorrhage probably occurred in the vitreous in all three cases, which may have resulted from damage to the ciliary vessels.

In the first case ultimate vision was 4/10. In the second the vision slowly returned to 1, partly. In the third case, because of secondary glaucoma, enucleation was advised. W. ZENTMAYER.

INTRAOCULAR FOREIGN BODY WITH BACILLUS WELCHII INFECTION. C. M. BEERY, Am. J. Ophth. **15:** 1022 (Nov.) 1932.

The patient was a robust man, aged 43. The eye was injured while he was cutting a nut off a motorcycle. Sixteen hours later there was active panophthalmitis with a stony hard tension. A roentgenogram showed a foreign body within the vitreous. A gas bubble was present in the anterior chamber. Attempts at extraction failed.

The following day the temperature was 102 F., and the patient was in great pain. The eyeball was enucleated. Bacillus Welchii was identified in cultures from the vitreous.

The author quotes Ridley's statement that, with severe pain, a rise of temperature and hemorrhage within twenty-four hours following a penetrating wound of the globe, a diagnosis of infection with Bacillus Welchii should be made without further evidence.

It is pointed out that French surgeons enucleate the eye in all such cases, while English surgeons resort to evisceration. In the foregoing case with ordinary enucleation, irrigation and immediate closure of the wound, healing was as prompt as is usually seen after the removal of an uninfected globe. W. ZENTMAYER.

AVULSION OF THE OPTIC NERVE AT THE SCLERAL OPENING AFTER A TRAUMATISM TO THE SUPERIOR ORBITAL MARGIN AND THE ROOT OF THE NOSE. P. SATANOWSKY, Arch. de oftal. de Buenos Aires **7:** 644 (Nov.) 1932.

After reviewing the causes found in thirty cases of avulsion of the optic nerve reported in the literature, the author reports a case in which the lesion followed an injury received when the patient was run down by a bus. There was also a fracture of the superior orbital margin and of the nasal bones. Five days after the injury, the fundus showed large, irregular white plaques, studded with hemorrhages. The disk was not distinguishable. At its site could be observed a circular, slate-colored space the size of the disk, surrounded by hemorrhages. The eye was blind. The fracture of the orbital margin and nasal bones was confirmed by roentgen examination. When the hemorrhages cleared up, a rupture of the choroid was found, with a retinal vessel proceeding from the margin of the opening. The opening later filled up with connective tissue. Roentgen examination of the optic foramen according to the method of Golawin showed no lesion at this site.

Papillary edema of the other eye was observed the first day, but disappeared three days after a lumbar puncture with drawing off of 40 cc. of bloody serum two days after the injury. C. E. FINLAY.

Lacrimal Apparatus

HISTOPATHOLOGIC CHANGES IN THE LACRIMAL SAC IN CHRONIC DACRYOCYSTITIS. A. VITTADINI, Arch. di ottal. **40:** 150 (April) 1933.

Twenty excised lacrimal sacs were examined grossly and histopathologically. The lumen varied from 30 by 15 mm. to less than 1 mm. in diameter. The epithelium showed all stages of mucoid degeneration,

resulting in the formation of mucous crypts where groups of degenerated cells had broken down. In certain sections these resembled true glands, but this appearance depended on the direction in which such crypts were sectioned. The walls of the sac were often infiltrated with leukocytes and lymphocytes, and their elastic tissue was often markedly degenerated. Vittadini believes that this loss of elastic tissue, combined with the pull of bands of connective tissue on the outer walls of the sac, is as important in producing ectasia of the sac as is the mechanical force of accumulated mucopus. The so-called polypi which were frequently found he believes are to be explained as part of the mucosa left between two mucous crypts rather than as protrusions into the lumen of hypertrophic processes. In only one sac was a true valve found at the nasolacrimal duct. He found no sacs with true glands in the mucosa and believes that the glands described by many authors were probably the mucous crypts described previously, which are due to mucoid degeneration of the epithelium. A bibliography and photomicrographs are included.

S. R. GIFFORD.

Lens

RADIATIONAL CATARACT. J. E. LEBENSOHN, Am. J. Ophth. **15:** 953 (Oct.) 1932.

The author and Gifford have corroborated the results of Aulamo and Rohrschneider, who first experimentally produced radiational cataract in adult animals.

Two patients with radiational cataracts were seen by the author. The first was a woman, aged 26, who had received intense roentgen treatment for acne at the age of 18. Six years later vision was 6/60 in the right eye and 8/30 in the left. Both lenses showed the characteristic postcortical opacities of cataract caused by roentgen radiation. The second patient was a man, aged 57. He worked as an iron molder from the age of 13 to 37. Vision was 6/240 in the right eye and 6/30 in the left eye. In each lens there were a posterior cortical opacity and in addition a well marked coronary cataract. W. ZENTMAYER.

THE EFFECT OF X-RAY AND RADIUM RADIATIONS UPON THE CRYSTALLINE LENS. C. A. CLAPP, Am. J. Ophth. **15:** 1039 (Nov.) 1932.

Fifty-four cases are gathered from the literature since 1903 in which lenticular changes resulted from the use of roentgen or radium therapy. (The abstractor reported a case of posterior polar opacity as a sequel to roentgen therapy following the removal of a sarcoma of the iris. More than five years have elapsed. The growth has not recurred, and the lenticular opacity has shown no increase of late.)

To the author it would seem wise to limit the use of this form of treatment to the more serious ocular conditions. It is possible, as shown by Bligvad, that if a filter of 3 mm. of lead is used, no opacities will result.

W. ZENTMAYER.

Methods of Examination

X-RAY DIAGNOSIS OF DOUBLE PERFORATION OF THE EYEBALL AFTER INJECTIONS OF AIR INTO THE SPACE OF TENON. E. W. SPACKMAN, Am. J. Ophth. **15:** 1007 (Nov.) 1932.

This method was suggested by Gasberger and Gauss. As employed by the author, it is as follows: After thorough anesthetization of the eye, the patient is instructed to turn it downward and inward; a point about midway between the superior oblique and external rectus is located, and the conjunctiva grasped, using sterile precautions. A curved cannula is then inserted between the conjunctiva and Tenon's capsule. Eight cubic centimeters of air is sufficient. When the eye is proptosed, owing to the presence of air posterior to the globe, and increased resistance is felt on the piston of the syringe, the injection is discontinued. The proptosis must be apparent, and this is the best guide as to the correctness of the procedure. If the conjunctiva bulges forward the needle is not within Tenon's space or there is too much leakage. In this case the test is of no value. The roentgenograms are made at leisure, preferably from several angles, as it is important to separate the shadow of the foreign body from that of Tenon's space. The layer of air below Tenon's capsule forms a band, which may be plainly seen in contrast to the denser bone tissue of the orbit, and by making pictures from various angles the relation of the particle to the capsule may be accurately demonstrated.

Three cases are reported, with illustrations.

W. ZENTMAYER.

CLINICAL RESEARCHES ON THE ANTIGENIC PROPERTIES OF UVEAL PIGMENT. S. RINALDI, Ann. di ottal. e clin. ocul. **61:** 268 (April) 1933.

Pigment suspensions were made up according to Woods' technic. The undiluted antigen contained the uvea of one beef eye and was added to 7.5 cc. of physiologic solution of sodium chloride. Dilutions of 1 : 10 and 1 : 25 of this antigen and a control antigen of beef serum with 0.5 per cent phenol were also employed. Intradermal tests were made with the whole antigen on 130 patients with various conditions involving the uveal tract. Of these tests 16.15 per cent were positive, the largest number being in patients with penetrating wounds and uveitis (26 and 25 per cent). Eighty patients without involvement of the uveal tract were tested, and 11.25 per cent of these tests were positive. With the 1 : 10 dilution, which was used in sixty-two cases of uveal injury or disease, 14.5 per cent of the tests were positive, while none were positive with the 1 : 25 dilution.

Only two cases of typical sympathetic ophthalmia occurred in the series, and these gave negative reactions to the whole pigment. There were six patients with slight signs of inflammation in the second eye, and two of these gave positive reactions.

In spite of the larger number of positive results in uveal disease, the positive tests obtained in some normal patients and the negative tests in sympathetic ophthalmia led the author to deny any diagnostic or prognostic value of the use of uveal pigment antigen.

S. R. GIFFORD.

INTRADERMAL REACTIONS WITH UVEAL PROTEINS IN UVEAL CONDITIONS. S. RINALDI and C. QUAGLIO, Ann. di ottal. e clin. ocul. **61:** 295 (April) 1933.

Suspensions of macerated uveal tissue (beef) were passed through a Berkefeld filter, 10 cc. of physiologic solution of sodium chloride being employed for each uvea. The protein content was free from pigment and contained 0.1137 per cent of protein. This solution was used for intradermal tests on fifty patients without uveal involvement and on one hundred and forty with uveal wounds or disease. In 4 per cent of the first series and in 7.85 per cent of the second series positive tests were obtained.

The patients with penetrating wounds gave the highest percentage (10) of positive tests, but these results had no relation to the outcome in these cases. Two cases of sympathetic ophthalmia and two of sympathetic irritation occurred in the series, all showing negative tests. The author concludes that sensitivity to uveal pigment is rare even in diseases of the uveal tract, at least so far as may be shown by intradermal tests, and that not much diagnostic or prognostic value may be given to such tests.

S. R. GIFFORD.

SIMPLE EQUIPMENT FOR PHOTOMICROGRAPHY OF THE CAPILLARIES OF THE LIPS AND CONJUNCTIVA. K. MÉSZÁROS, Orvosi hetil. **76:** 975 (Oct. 29) 1932.

According to Mueller light might be thrown on the general capillary system by studying the surface of the skin and the mucous membranes. The mucous surface of the lips and the conjunctiva lend themselves best to researches of this kind but particularly to photomicrographic studies. The apparatus of Bock, very well suited as it is for the photomicrography of the capillaries of the conjunctiva, is said to fall short of the requirement of easy use. Mészáros has therefore improvised a simpler apparatus, consisting of a Reichert microscope mounted on a tripod. A photomicrographic apparatus of Leitz is attached to the microscope. The microscope is movable in any direction and assumes a nearly horizontal direction in examination of the conjunctiva. A green filter is inserted in front of the luminous rays. A photomicrograph of the capillaries of the conjunctiva is given.

N. BLATT.

Ocular Muscles

DEMONSTRATION OF "COUNTERROLLING" OF THE EYES BY MEANS OF STRUYCKEN'S APPARATUS. R. CAUSSÉ, Arch. d'opht. **50:** 353 (May) 1933.

Counterrolling is defined as the movement of the eyes in the opposite direction to the head in an attempt to maintain the primary position in space. For instance, when the head is inclined to the shoulders, or the body is inclined, the eyes only partly follow this movement. This phenomenon has been known for many years and was of purely academic interest until it was shown by Breuer to be a reflex arising in the labyrinth. The control of these movements was experimentally shown to be situated in the utricle by Versteegh, whose work has been confirmed

by Maxwell, Tait and MacNally. As a result of these studies a new interest has resulted in counterrolling as a means of investigating the labyrinth. It has been applied to various conditions, but the measurements have been so widely different that its value is questioned. This is due to the difficulty of performing the test, and for this reason the author describes a simple accurate procedure based on the apparatus of Struycken. This instrument and its use are described.

S. B. MARLOW.

Operations

OPERATION FOR EPICANTHUS TARSALIS. E. OLAH, Klin. Monatsbl. f. Augenh. **90**: 233 (Feb.) 1933.

The author reports a case of tarsal epicanthus which could not be made to disappear except by lifting the skin in the direction of the nasal termination of the brow. This differed from previous experience in dealing with palpebral exophthalmos. Olah excised an elliptic piece of the skin at the medial portion of the brow, between the supraorbital foramen and the nasofrontal suture. The excised piece corresponded to the folds and was larger than is customary in methods in which the site for the excision is chosen between the two nasal canthi. Correction of the epicanthus resulted, with an invisible scar, which formed an inconspicuous continuation of the brow.

K. L. STOLL.

Orbit, Eyeball and Accessory Sinuses

SEQUELAE OF THROMBOPHLEBITIS OF THE CAVERNOUS SINUS. F. KOLLER, Arch. f. Ophth. **130**: 64 (May) 1933.

The author studied seven cases of thrombophlebitis of the cavernous sinus clinically and anatomically. In six of the seven cases suppurative inflammatory lesions in the wall of the carotid artery with consecutive arterial thrombosis were found; they accounted for the ophthalmoscopic picture of embolism of the central artery, which had been diagnosed in one case intra vitam. The nerve lesions in thrombophlebitis of the cavernous sinus could also be explained by extension of the inflammatory process to the cranial nerves within the sinus.

P. C. KRONFELD.

CHLOROMA OF THE ORBIT. N. BLATT, Miscarea med. **5**: 495, 1932.

The etiology and histopathogenesis of chloroma stand in need of elucidation. Some authors feel that this peculiar tumor is a focal symptom of a generalized systemic disease; i. e., it represents a biologic variety of leukemia. Other authors state that the pathologic analogies existing between the syndrome of chloroma and that of leukemia are insisted on unduly, as the chloroma is to be considered a variety of sarcoma.

Clearing up the pathologicoclinical aspect of chloroma is of course much more difficult because cases of it are fortunately rare. The number of cases of orbital chloroma, including the case reported here, total seventy-five.

A girl, 3 years of age, showed marked exophthalmos on the right; the eyeball was deviated toward the temporal side and upward. The pupil was widely dilated and did not react either to light or in convergence. On digital palpation uneven tumor masses of hard consistence could be felt above and below the eyeball.

The left eyeball lay before the eyelids and was almost completely luxated. It was surrounded by greenish-brown tumor masses and was embedded in them.

The roentgenogram showed infiltration of, and periosteal radial formation of new bone on, the frontal bone, and destruction of the anterior portion of the body and of the right greater wing of the sphenoid bone, as well as of the posterior portions of the ethmoid bone and also the left malar bone. The patient died eight weeks after examination.

This case history is at once suggestive of chloroma.

The roentgenogram and the blood count are of the greatest value in the diagnosis of chloroma. The blood count does not offer a strict classification of the type of the chloroma. The degree of the alteration of the blood is dependent not so much on the size and extension of the tumor as on the length of time that it has been present and on the location of the metastases. The blood count has to be considered only as a symptom and as a malignant symptom in far advanced cases. Chloroma is a peculiar multiple malignant tumor which attacks especially the bones, thus causing disease of the bone marrow and blood-forming organs.

Chloroma ought to be separated from leukemia and assigned a particular place in pathology. Chloroma is considered to be bone sarcomatosis occurring in subjects with defective hematopoietic systems. It seems that some congenital factor is the basis of the incompetence of the blood system which causes bone sarcomatosis to assume the form of chloroma.

N. BLATT.

Pharmacology

INFLUENCE OF p_H ON THE PHYSIOLOGIC ACTIVITY OF COCAINE SOLUTIONS. J. RÉGNIER and R. DAVID, Compt. rend. Soc. de biol. **114**: 977, 1933.

Cocaine hydrochloride, when mildly alkalinized, shows a marked increase in anesthetizing power but does not possess keeping qualities. Hence cocaine solutions should be made neutral or slightly acid.

J. E. LEBENSOHN.

The Pupils

THE ARGYLL ROBERTSON PUPIL. H. H. MERRITT and M. MOORE, Arch. Neurol. & Psychiat. **30**: 357 (Aug.) 1933.

Merritt and Moore review recent anatomic and physiologic studies on the pupillary pathways and find therein an explanation for the Argyll Robertson pupil. The Argyll Robertson pupil has six characteristics: 1. The sensitivity of the retina is not impaired, i. e., the light sense is not materially affected. 2. The pupils are miotic. 3. The pupils remain constant in size in spite of changes in the intensity of the illumination. 4. They contract actively in accommodation convergence. 5. They dilate imperfectly in reaction to atropine. 6. They do not dilate when painful stimuli are applied to the skin.

The sensitivity of the retina implies that the optic visual pathways are relatively intact. Miosis may be due to either paralysis of the sympathetic or stimulation of the parasympathetic nerve supply. Since the miosis persists for years it is unlikely that it is due to stimulation of the parasympathetic nerve supply. The failure of the pupil to react to light can be explained only as due either to an interference with the afferent fibers from the retina to the nucleus of the third nerve or to a paralysis of the innervation of the sphincter muscle. That the latter is not the cause is proved by the prompt contraction of the pupil during accommodation convergence. Failure of the pupils to dilate perfectly in reaction to atropine is characteristic of paralysis of the sympathetic innervation of the eye. Finally, the failure of the pupils to dilate in response to painful stimuli must be due to interference with the sympathetic supply to the dilator muscle.

Recently, Beattie and his co-workers (*Proc. Roy. Soc., London, s.B.* **106:** 253, 1930; *J. Anat.* **66:** 283, 1932) localized a center in the posterior portion of the hypothalamus which controls the true sympathetic nervous system. They showed that fibers running from this center enter the anterior colliculus by passing ventrally to the posterior commissure. From there some of the fibers arch ventrally to enter the medial lateral portion of the midbrain and descend in the reticular formation of the pons and medulla. Others arch ventromedially to run in the homolateral and contralateral posterior longitudinal bundle. They found that the pupillodilator fibers were uncrossed and were in connection only with the homolateral cervical sympathetic center. Thus, it can be seen that in passing ventrally these fibers are in close connection with the pupillomotor fibers.

From the facts outlined it may be seen that a single lesion can cause all the phenomena of the Argyll Robertson pupil. This lesion would have to be in the tegmentum ventral to the posterior commissure, where the light reflex fibers are coursing ventromedially to the oculomotor nucleus and where the sympathetic fibers are coursing ventromedially to the posterior longitudinal bundle and ventrolaterally to the reticular formation.

These facts prove that the Argyll Robertson pupil can be produced only by injury to both the afferent pathway and the sympathetic fibers. Also, if the phenomena are to be explained on the basis of a single lesion, this must be at a point where the two pathways converge.

The afferent fibers for the light reflex originate in the retina and decussate in the optic chiasm in the same manner as the visual fibers. They leave the visual fibers, however, at about the region of the external geniculate body and go with the brachium of the superior corpora quadrigemina toward this body. They pass to the posterior tip of the superior colliculus, and some cross in the dorsal portion of the posterior commissure, while the remainder arch ventrally toward the oculomotor nucleus. Ranson ("The Pathway of the Pupillary Light Reflex," read at the Fifty-Ninth Annual Meeting of the American Neurological Association, Washington, D. C., on May 9, 1933) found that constriction of the pupils was obtained when the brachium of the superior quadrigeminal bodies was stimulated and when the posterior commissure was stimulated. Except for a small tract in the pretectal region between the posterior commissure and the oculomotor nucleus,

where stimulation produces constriction of the homolateral pupil only, stimulation in the region of the anterior colliculus produced dilatation of the pupils. Ranson's work shows that the light reflex fibers which cross in the optic chiasm recross in the posterior commissure. This is of great importance, because destruction of the pathway of the light reflex after these fibers have recrossed would cause unilateral Argyll Robertson pupil. No theory of the localization of the lesion producing the Argyll Robertson pupil is valid unless it also explains the unilateral occurrence.

The authors insist, and probably rightly so, that the term "Argyll Robertson pupil" should be strictly limited to cases in which all the pupillary phenomena just outlined are present. If there is impairment of vision it is obvious that a diagnosis of Argyll Robertson pupil cannot be made with certainty. There are many pupils which do not react to light but are active in accommodation convergence which are not true Argyll Robertson pupils. The best example of this is the blind eye.

After reviewing the records of 749 cases of neurosyphilis the authors found that the Argyll Robertson pupil had been observed in 38 per cent. They agree with Adie (*Brit. M. J.* **2:** 136, 1931) that the true Argyll Robertson pupil is practically an infallible sign of syphilis of the nervous system. They point out that the only other lesion which may cause this typical syndrome is an infiltrative lesion, such as a glioma of the anterior colliculus. F. H. ADLER.

Physiology

LIGHT SENSE. P. W. COBB, Am. J. Ophth. **15:** 917 (Oct.) 1932.

Tests for dark-adaptation have proved the most satisfactory methods of studying light sense. The author gives the following summary:

1. The term "light sense" taken broadly has as many meanings as there are possible methods of measuring it.

2. This is due in part to the fact that any visual object, or test object, requires at least four terms for its complete description.

3. These are: first and second, two photometric terms, the brightness of the figure and the brightness of the background, which often may conveniently and adequately be expressed as the brightness level and the brightness difference; third, the size of the object, best expressed as its angular extent in the visual field, and fourth, the time of action of the object.

4. The fact that any or all of these terms or dimensions may be varied by many stages gives rise to a legion of possibilities from which a test object must be selected.

5. Since the eye works by means of a medium, the light, which changes enormously from time to time, it may be said that an important function of the eye is to ignore changes in this medium, which are irrelevant to the interpretation of objects seen.

6. Tests of light sense which have been tentatively applied in clinical ophthalmology are the brightness difference test and the dark-adaptation test. Experience with the former has, on the whole, been unpromising by reason of the wide and irregular variability of the results.

7. The subject of dark-adaptation leads directly to the fact that there are two distinct phases of vision, corresponding respectively to vision under full light with discrimination of colors and to vision under dim light without color discrimination. The former phase has been theoretically referred to the activity of the cones (with the possible participation of the rods), and the latter to the activity of the rods alone.

8. Regardless of theory, the dark-adapted eye works in the second of these two phases, and studies on dark-adaptation should take full account of the known laws of scotopic vision.

9. The literature on dark-adaptation shows a confusing number of different technics and ways of stating results. Certain essential details of method and essential precautions are enumerated, with a view to uniformity and to the elimination of variability in the results due to non-essential factors.

W. ZENTMAYER.

THE SPECTRAL SENSITIVITY OF THE SUN FISH AS EVIDENCE FOR A DOUBLE VISUAL SYSTEM. H. GRÜNDFEST, J. Gen. Physiol. **15**: 507, 1932.

By an appropriate apparatus which determines the amount of light energy from different portions of the spectrum which will just produce a visual orienting response in the sunfish to the movement of a pattern, Gründfest finds that this function differs with dim and bright illumination. This difference, he believes, indicates that the eye of the sunfish is a physiologic duplex visual system and parallels the known anatomic distinction between the rods and the cones.

F. H. ADLER.

RÔLE OF FILTRATION IN THE MAINTENANCE OF THE INTRAOCULAR PRESSURE. JOHN PARSONS, Quart. J. Exper. Physiol. **23**: 51, 1933.

Parsons reviews the evidence supporting the dialysate theory of the formation of the aqueous. He points out that the intra-ocular fluid is comparable in all respects to normal tissue fluid, i. e., to the lymph of other tissues.

The chemical and physical properties of the aqueous can be explained according to the laws of dialysis, and there is no satisfactory evidence as yet to prove that secretion plays a part in the formation of this fluid. A dialysate is a fluid in equilibrium with the parent fluid from which it was formed—in this case, the blood.

There is ample evidence that the aqueous is not a stable fluid. Every change in intra-ocular pressure, such as is caused by simple closing of the lids, and every change in capillary pressure within the eyeball will upset the equilibrium between the aqueous and the blood. It is necessary to postulate, therefore, a mass movement of fluid within the eyeball, even though this may be very small. In this way the theory of dialysis and the older theory of filtration become reconciled.

No new evidence is given to support this view, which has been advocated by earlier authors (Friedenwald and Pierce: ARCH. OPHTH **7**: 538, 1932).

F. H. ADLER.

Refraction and Accommodation

MYOPIA IN RELATION TO THE GENERAL BIOLOGIC LAWS. A. V. BARRIERE, Arch. de oftal. de Buenos Aires **7:** 515 (Sept.) 1932.

This paper is a study from a biologic point of view of the prevalence of myopia. After eliminating the pathologic varieties, the author considers the problem at great length from different aspects. He found that the incidence of the condition corresponds to the general laws of inheritance and natural selection observed in connection with other anatomic characteristics. The greater proportion of myopia found among children in advancing school years has no relation to eye work and depends on the initial inherited refraction and the natural body growth.

C. E. FINLAY.

Retina and Optic Nerve

A CASE OF DIABETIC RETINITIS. A. B. ZORAB, Brit. J. Ophth. **17:** 41 (Jan.) 1933.

The interest in this case was the picture presented by the fundus. The patient was a man aged 22. The sugar content of the urine varied from 13 to 40 grains (0.845 to 2.6 Gm.) per ounce (31.08 Gm.).

"The blood examination failed to reveal any marked divergence from the normal, and its color when drawn from a pricked finger was quite satisfactory."

The general fundus reflex was normal, slightly blurred perhaps by the edema of the retina. The vessels showed up in a startling fashion, silvery white, bright and distinct; even the smallest twigs could be seen quite easily by the direct method. An artery could be followed from one of the main branches until it broke up into a veritable network, and a vein could be traced back from this network to the main veins. In three separate parts of the fundus in each eye an anastomosis between two small arteries could be seen with absolute distinctness. There were two such anastomoses between branches of the inferior and temporal retinal arteries and one anastomosis between branches of the inferior and nasal retinal arteries.

The case was not one of lipemia retinalis. The report is illustrated.

W. ZENTMAYER.

THE SENSITIVITY OF THE CORNEA AFTER CERTAIN OPERATIONS FOR RETINAL DISPLACEMENT. E. SPITZER, Arch. f. Opht. **130:** 41 (May) 1933.

After so-called demarcation (*Abriegelung*) operations for retinal detachment, the sensitivity of the corresponding sector of the cornea remains impaired for several months. This disturbance lasts about eleven months after the Guist operation (multiple trephine) and for about eight months after Lindner's undermining operation. The hypesthetic sector is very small after microcoagulation, larger after undermining and largest after the Guist operation. The recovery of sensitivity starts from the central portion of the affected sector and spreads toward the periphery.

P. C. KRONFELD.

NEUROFIBRILLITIS RETINAE (E. JENSEN): REPORT OF THREE CASES. W. RIEHM, Klin. Monatsbl. f. Augenh. **90:** 154 (Feb.) 1933.

Neurofibrillitis of the retina is identical with "retinochoroiditis juxtapapillaris," described by E. Jensen in 1908. The disease is manifested by blurred vision and a slightly prominent, diffused, whitish infiltration, round or oval, located near the disk or extending into its tissues. The infiltration comprises the innermost layers of the retina and frequently hides the retinal vessels. An absolute and irreparable defect of vision corresponding to the retinal fibers crossing the infiltration is the most characteristic symptom. The defects are usually sector-shaped or arc-shaped; they comprise the center of fixation only if the macula is involved. The blurred vision observed at the onset is caused by fine opacification of the posterior portion of the vitreous, chiefly that situated in front of the infiltration. The atrophic scar, resulting in many but not in all cases, involves the pigment layer and often the inner layers of the choroid. The choroid beneath the scar hardly ever disappears completely, and its larger vessels usually remain intact, in contrast with disseminated choroiditis. The patients are mostly healthy persons in the second and third decades of life. The disease is rarely bilateral, nor does it show progressive tendencies. When associated with intercurrent general diseases, however, it is prone to recur in the neighborhood of the original focus. The cause of the defect of the field of vision remains unsettled. Jensen, Zentmayer and Ormond attribute it to disturbances of the circulation of the providing artery; van der Hoeve, Rönne and others, to a lesion of the retinal fibers crossing the focus.

The author reports in detail three cases, two in women, aged 20 and 18, and one in a man, aged 25, and draws the following conclusions: The primary seat of the disease is in the retina (contrary to the opinion of Schieck, Gilbert and Rönne), and tuberculosis is the main etiologic factor. A general reaction followed the smallest doses of old tuberculin in Riehm's three cases. Multiple sclerosis and syphilis may be mentioned as etiologic factors, and grip, erysipelas and injury are contributing causes.

K. L. STOLL.

MODELS FOR EXPERIMENTS ON THE MECHANISM OF DETACHMENT OF THE RETINA. K. LINDNER, Klin. Monatsbl. f. Augenh. **90:** 289 (March) 1933.

Lindner reports in detail on the construction of his models used in demonstrating detachment of the retina, which he showed at the meeting of the German Ophthalmological Society at Leipzig in 1932. A report on the models of the vitreous and on experiments performed with their aid is appended.

By means of one of these models, Lindner explains the mechanism of detachment of the retina to patients undergoing treatment. The models show the detrimental influence of the motion of the globe on the detached retina. Another type of model permits observation of the process of detachment after a rupture occurs. The third type, the models of the vitreous, explains the forces responsible for the rupture. A round glass flask after Schott, Jena, form D, of 500 Gm. content,

with a neck 40 mm. in width, is used for the first two types of models. The inner surface is covered by rotation with an equally distributed layer of a 5 per cent solution of pyroxylin, which will dry within two minutes under compressed cold air. A second layer consisting of a 2 per cent solution of pyroxylin and containing powdered alum is added, which produces a silvery, easily visible lining. The flask is filled with water on the following day and closed with a rubber stopper after the air is eliminated, and a large hole is torn in the lining with a metal spatula. The fluctuation of the membrane, representing the detached retina, can be demonstrated by rotation of the flask.

The second type, showing the influence of the hole on the origin of the detachment, is prepared with only one layer of a 2 per cent solution of pyroxylin with aluminum, which will partly adhere to the flask. Only a small hole is torn in the membrane.

For the third type, a mixture of gelatin and glycerin, physically resembling the vitreous, is used. The origin of Gonin's flap-shaped and horseshoe-shaped ruptures and of total detachment of the retina without preceding trauma can be demonstrated by a jerking motion of the model around its center. Eyes threatened with imminent detachment should be trained to acquire a staring gaze. This is facilitated by the use of spectacles with strong marginal astigmatism. K. L. STOLL.

BILATERAL ATROPHY OF THE OPTIC NERVE ASSOCIATED WITH UNILATERAL SPHENOID PNEUMOSINUS. T. BENDESCU, Rev. stiint. med. **21**: 570, 1932.

A girl, 24 years of age, had for one year remarked a slow but progressive decrease in the vision of both eyes. The case history did not give any clue to the etiology. Vision in the right eye was 1/20 and that in the left eye, 1/50. The visual field of the right eye showed concentric contraction up to 10 degrees. The visual field of the left eye could not be determined. The reactions of the pupils were normal. There was total simple atrophy of both disks. The physical examination gave negative results. The roentgenogram showed the base of the brain forced upward. This was due to dilatation of the pneumatic right sphenoid sinus. The optic nerve canal showed a kidney-shaped deformation and marked narrowing. The left sphenoid sinus and the ethmoid sinuses were free from pathologic changes. The increase in size of the right sphenoid sinus had brought about the displacement of tissue which was responsible for the alteration of the optic nerve canal and the atrophy of the optic nerve.

The dilatation of the right sphenoid sinus had originated from the chronic hypertension of the air contained in the sinus. N. BLATT.

IS "RETROBULBAR NEURITIS" AS OFTEN DUE TO DISEASE OF THE NASAL ACCESSORY SINUSES AS IS GENERALLY HELD? N. BLATT, Tenth Cong. Rumanian Soc. Otorhinolaryng., Bucharest, Oct. 29-30, 1932.

As an etiologic factor in retrobulbar neuritis disease of the nasal accessory sinuses is still enormously overrated. On the other hand, it is evidenced by exact and carefully gathered and interpreted statistics that retrobulbar neuritis is more frequently due to multiple sclerosis than

to disease of the nasal accessory sinuses. A statistical table of forty-four cases of retrobulbar neuritis which I have had the opportunity of seeing in the course of fifteen years and observing for some length of time is given. In this table appear the rhinologic diagnosis of the involvements of the nasal accessory sinuses, the instances in which the neurologist was able to find evidence of multiple sclerosis, and the instances in which rhinologic operations were performed.

Owing to the protracted observation of the cases these statistics, brief as they are, can be given credit for their objectiveness. The table shows that the number of cures obtained in cases in which operation was performed was not larger than the number obtained in cases in which no operation was performed. Nor was the course of the disease influenced by operation.

In retrobulbar neuritis operative treatment of the concomitant disease of the nasal accessory sinus may, of course, but not necessarily, assist in the cure of the nervous process. In order to influence the optic nerve process, operation should be done only in the presence of a purulent or seropurulent inflammation of a nasal accessory sinus and only when multiple sclerosis is absent. In this respect the ethmoid cells, the sphenoid and frontal sinuses and lastly the antrums of Highmore, in the order mentioned, should be borne in mind as common sites of disease causing retrobulbar neuritis. One should not, however, assume that a hypertrophy of the nasal mucous membrane, a polyp or a deflection of the septum which happens to coexist is the cause of the retrobulbar neuritis, or expect a cure of the diseased optic nerve from an operation done for one of these conditions.

It should, moreover, be taken into consideration that multiple sclerosis in the first stages of its appearance may be located in the optic nerve, so that retrobulbar neuritis proves an important, indeed, often the only, early symptom of multiple sclerosis.

That opening of the nasal accessory sinuses, even if they show only slight lesions, may in some cases be followed by improvement of the diseased optic nerve is mentioned only to point out that retrobulbar neuritis due to multiple sclerosis is known to improve in some cases spontaneously and quickly. N. BLATT.

Trachoma

FILTRABILITY OF THE VIRUS OF TRACHOMA. U. LUMBROSO and P. THYGESON, Arch. Inst. Pasteur de Tunis **22**: 178, 1933.

Experiments on the transmissibility of trachoma must be limited to man, the anthropoid apes and the Algerian baboon because it is doubtful whether any other species is susceptible. This consideration renders most of the previous work on the filtrability of the virus of trachoma questionable. The findings of Lumbroso and Thygeson were negative, thus confirming the original conclusions of Pfeiffer and Kuhnt and Hess and Römer. J. E. LEBENSOHN.

Tumors

MALIGNANT TUMORS OF THE LACRIMAL SAC. J. A. SEÑÁ, Arch. de oftal. de Buenos Aires **7**: 603 (Oct.) 1932.

After a brief consideration of the normal anatomic and histologic characteristics of the lacrimal sac and a review of the present knowledge of the rare tumors of this organ, both benign (cysts and polypi) and

malignant (carcinomas and sarcomas), the author refers to two cases seen in consultation. In one case, at operation for an apparent mucocele a tumor was discovered which proved to be a cylindroma. In the second case, in which the patient was operated on for an apparent dermoid cyst, a sarcoma was discovered. The author finally reports in great detail a case of his own of recurrent carcinoma. The original tumor had been excised by another ophthalmologist during an operation for a supposed mucocele several months before Señá saw the patient. This patient was treated by electrocoagulation, followed by applications of radium. A complete cure resulted. No recurrence had taken place eight months later. A plastic operation was indicated for cosmetic purposes.

C. E. FINLAY.

CHOLESTEATOMA OF THE ORBIT: REPORT OF TWO CASES. F. PINKUS, Klin. Monatsbl. f. Augenh. **90:** 145 (Feb.) 1933.

The occurrence of cholesteatoma of the orbit in two men, aged 43 and 46, is reported. One of them noticed the disturbance when double vision developed as a result of displacement of one eye; the other man, when painful swelling of one eye set in. The cause remained unknown and may have to be traced to congenital "anlage." Repeated attacks of inflammatory and degenerative processes may have led to the destruction of the capsule of these tumors, which is generally missing. The result was the formation of cholesterol in the organized and proliferated tumor, which produced a typical anatomic appearance: granulomatous tissues with many cells and some giant cells, pseudoxanthoma cells, hemin, hematin and cholesterol crystals. There were no bacteria. The periosteum was degenerated, exposing the brittle and thinned orbital bone, in which cavities were found, the size of a walnut in one case. Histologic examination of the marginal portions of these tumors failed to reveal whether they were formed of changed periosteum.

The orbital cholesteatomas belong to the cystic tumors; they may be related to the dermoid cysts, and they should be termed "pseudocholesteatomas of the orbit" if considered as a separate group.

K. L. STOLL.

Uvea

THE DIAGNOSIS OF THE IRIDO-CILIARY TUBERCULOSIS. H. LAGRANGE, Brit. J. Ophth. **17:** 679 (Nov.) 1933.

This is the author's introduction to his report on the direct and differential diagnosis of tuberculosis of the iris and ciliary body to the International Congress of Ophthalmology in Madrid in 1933.

Lagrange first takes up the early view that ocular tuberculosis is a secondary lesion supposedly due to superinfection. A fundamental idea is derived from this view, namely, that iridociliary tuberculosis even when occurring as an isolated sign in an organism appearing to harbor no other localization of tuberculous infection is derived from a primary site, which demands initial study and examination. Definite proof of the presence of primary infection is required for diagnosis. As to pulmonary signs, he agrees with Rist "that pulmonary forms of tuberculosis, on the one hand, and extra-pulmonary manifestations, on the

other, form two distinct groups, since they present more than one contrasting character and exhibit a singular sort of balance."

It goes without saying that the digestive tract, bones, joints and lymphatic system should also be thoroughly examined. The author stresses the importance of a study of the cardiovascular system. The tuberculous heart is characterized by tachycardia, microcardia and hypotension. When the humoral test proves the presence of tuberculosis, the etiologic diagnosis of the ocular lesion is not established. He asks the question "Is not the occurrence of a cuti-reaction positive to tuberculosis entirely a normal sign?"

Three special methods, however, appear to be highly promising: tuberculin diagnosis applied, according to the given case, either in the form of test treatment or in the form of the homeotopic technic of Marc Weiss; microleukocytic culture as applied by Loewenstein; search for the ultravirus of tuberculosis by the intraglandular inoculation of guinea-pigs. The morphologic diagnosis of iridociliary tuberculosis is merely referred to.

W. ZENTMAYER.

IRITIS IN CHRONIC SPONDYLARTHRITIS ANKYLOPOETICA: REPORT OF CASES. EBERHARD KUNZ, Klin. Monatsbl. f. Augenh. **91:** 153 (Aug.) 1933.

Seven cases are reported of iritis in chronic inflammatory stiffening of the spine—spondylarthritis ankylopoetica. The author points out that this syndrome is more frequent than hitherto suspected, and that this type of iritis is of a rheumatic nature. Hence, the opinion of those internists who consider spondylarthritis ankylopoetica as chronic arthritis of the spine is corroborated. Therefore, roentgenograms of the spine, which shows rheumatic changes earlier than do the extremities, may be valuable in proving the presence of arthritic changes. In only four of the seven cases reported was there an undoubted anamnesis of rheumatism, and in two of them, of stiffening of the joints of the extremities. Early stages of arthritis of the spine may be discovered by a roentgenogram taken of a patient with iritis of an uncertain or suspected rheumatic nature. Noninflammatory spondylitis in connection with rheumatic iritis deserves no consideration in this matter.

K. L. STOLL.

THE SO-CALLED "IRIS DIAGNOSIS." F. SALZER, München. med. Wchnschr. **80:** 1146 (July 21) 1933.

Frequently reports of the ability to diagnose bodily disorders by means of alterations of the irides have crept into the literature. There has been much antagonism by the general practitioner against this type of occultism, and justly so. The author cites cases reported and goes into detailed explanation to demonstrate their inaccuracy. He concludes:

1. It is impossible to make a diagnosis of a bodily disease by reference to the condition of the iris.

2. The counterevidence against these collections of evidence can never be proved through the theoretical arguments or references to true or alleged cures, but only through practical investigations.

L. L. MAYER.

TUBERCULOSIS OF THE UVEA. G. HORAY, Orvosképzés 12: 26, 1932.

This paper is a comprehensive account of the older as well as the modern views of the pathology of uveal tuberculosis. Since the time of Michel it has been known that tuberculosis of the iris is not a local disease but the most important, often even the only, phenomenon of a systemic tuberculous infection.

Formerly tuberculosis was made light of as a causative factor of uveitis; at present it is often overrated. The etiology of uveitis can sometimes be cleared up only by study of the clinical picture.

Positive or negative pulmonary findings do not argue either for or against the tuberculous etiology of the ocular disease. In the first years of life the proliferative forms of tuberculous uveitis occur in association with conglomeration and caseation; between the twentieth and thirtieth years of life there is an increase in the incidence of exudative iridocyclitis both with deposits and with predisposition toward glaucoma, and in elderly people there are mostly progressive processes hardly distinguishable from other forms of uveitis.

It is still open to question whether it is the tubercle bacillus alone or its toxins, too, which can cause the disorder of the uvea. The classification of Fuchs in evolved and atypical tuberculosis of the uvea holds true from the didactic point of view. The relation of myopia to tuberculous disseminated choroiditis still remains a moot point. According to Werdenberg and Krueckmann this much has been safely established: Uveal tuberculosis is always a metastasis of generalized tuberculous disease; the lungs are very often affected, and first and foremost the tuberculous lymph nodes of the mediastinum are predisposed to spread metastases to the eyeball. The course of ocular tuberculosis depends on the state of the equilibrium between infection and immunity.

N. BLATT.

Vision

THE OCCURRENCE OF NIGHT BLINDNESS IN MEN AND WOMEN. G. JOACHIMOGLU and G. LOGARAS, Deutsche med. Wchnschr. **59:** 1043 (July 7) 1933.

Birnbacher has shown that hemeralopia occurs preeminently in males and seldom in females after the age of puberty. The authors found only ninety-four cases of essential hemeralopia recorded in the literature. A curve of the age incidence of these cases is shown, demonstrating Birnbacher's contention. The authors are of the opinion that the condition is due to a deficiency in vitamin A as shown by experiments on animals. They believe that the explanation is dependent on the amount of fat deposits. The female is much better equipped. This difference has also been found in the fat deposits of the cow and the steer.

L. L. MAYER.

Visual Tracts and Fields

FIELDS OF VISION IN INTRACRANIAL LESIONS. H. M. TRAQUAIR, Brit. M. J. **2:** 229 (Aug. 5) 1933.

Quantitative perimetry is carried out by serial stimuli varying from a 1 mm. test object to a strong electric light in order to elicit the "characters" of field defects. These characters are: (1) position, (2) shape,

(3) size, (4) intensity, (5) uniformity, (6) margins, (7) behavior, or course, (8) presence of proportion or disproportion and (9) presence of congruity or incongruity.

It is evident that all hemianopias due to suprageniculate lesions must be congruous. The one exception to this rule is due to the fact that the temporal and nasal fields are not normally coextensive, the temporal field being the larger by a crescentic area about 30 degrees wide at its widest part. This area corresponds to the nasal periphery of the retina, to the inner side of the visual path and, posteriorly, to the most anterior part of the occipital cortex. It is possible in a suprageniculate hemianopia for the temporal 30 degrees of the binocular field to be affected alone or to be spared in an otherwise congruous homonymous hemianopia.

The author divides the visual pathway into a suprageniculate part, including the radiations and the cortex, and a subgeniculate portion, which may be divided into subchiasmal, chiasmal and suprachiasmal sections, with characteristic field changes for each part. Since it has been shown by Cushing that tumors of the temporal lobe produce incongruous homonymous hemianopic defects, the author thinks that it is the optic tract (and not the radiations) which is affected by a tumor in this location. A case of Kravitz' is quoted which supports the foregoing view, and Horrax and Putnam, who showed that tumors of the temporal lobe were characterized by the frequency of incongruity and of upper quadrantal defects, and that sparing of the central area was relatively rare in such cases, are also quoted to support the foregoing view.

W. F. DUGGAN.

Therapeutics

FEVER THERAPY IN TABETIC ATROPHY OF THE OPTIC NERVE. B. FLEISCHER, Klin. Monatsbl. f. Augenh. **90:** 335 (March) 1933.

Careful treatment with malarial infection in eight women showed not only no adverse results, but a decided improvement of the vision after observations continued over long periods. Discontinuation or retardation of the progress of the atrophy is frequently all that can be expected in fulminant cases. Quinine suffices to reduce the fever, if desired. Malaria toxin seems to act with especial intensity in metasyphilitic conditions.

The author used injections of sulphur in olive oil in two of his patients, a prompt rise of the temperature resulting. This preparation, to which benzocaine (anesthesin) may be added to allay the lancinating pain, was well tolerated. Its effect may be increased by bismuth and it promises to replace eventually the malaria therapy. Fleischer suggests the use of the parenteral sulphur therapy by his confrères for the purpose of gaining more reliable data. The injections should be made in the evening, so as not to disturb the patient's rest, as the temperature will not rise within ten hours.

K. L. STOLL.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

PARKER HEATH, M.D., *Secretary*

Milwaukee, Wis., June 14, 1933

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EFFECT OF TRYPARSAMIDE ON THE EYE. DR. N. K. LAZAR, Chicago.

This article appeared in full, with the discussion, in the February, 1934, issue of the ARCHIVES, page 240.

THE CILIARY PROCESSES AND THEIR RELATION TO INTRA-OCULAR OPERATIONS. DR. ALGERNON B. REESE, JR., New York.

Increase in volume and alteration in position of the ciliary processes lead sometimes to their incarceration and even excision when operations are performed on the iris. The conditions which tend to alter the ciliary processes and thereby render them liable to involvement in intra-ocular operations are: (1) advancing age, (2) perforation of the globe, (3) glaucoma and (4) congenital malplacement.

1. *Advancing Age.*—It has been demonstrated by Hess that the length and breadth of the ciliary processes are strikingly less in the new-born than in adults, and that these dimensions increase progressively with age.

2. *Perforation of the Globe.*—This is followed by an effusion of fluid into all the intra-ocular tissues, the edema in the ciliary body and processes being especially marked. Eyes enucleated immediately following a laceration or a perforation of the cornea or of the limbus commonly show an increase in volume of the ciliary structures, and the same observation is made following operative incisions that empty the anterior chamber.

3. *Glaucoma.*—In cases of acute and chronic congestive glaucoma, the volume of the ciliary body, particularly in its anterior portion and its processes, is increased by edema. In cases of chronic glaucoma of long standing, however, it is common to see a small, atrophic ciliary body and small, atrophic processes, although even in these cases Priestley Smith considered that there is evidence to support the contention that in the earlier stages they were edematous.

4. *Congenital Malplacement.*—The ciliary processes sometimes arise from the posterior surface of the iris, in its periphery, instead of from the normal site on the corona ciliaris. When they encroach on the posterior surface of the iris as described, they lend themselves to incarceration and even excision when operations are performed on the

periphery of the iris. This is especially true in the case of the trephine operation. If the ciliary processes are as vulnerable as one is led to believe their incarceration or excision may lead to iritis. The processes are largest from edema in congestive glaucoma, and it is just in this type of condition that iritis is most frequently seen following the trephine operation.

PRACTICAL LENSES. A TRIAL SET. DR. EDWARD JACKSON, Denver.

Trial sets should be especially planned for those who use them. Plano lenses with wire trial frames furnish the best and most convenient sets. Twenty pairs of lenses will make all the combinations needed to correct any condition likely to be encountered. Single lenses of each strength can be combined in order to correct the two eyes for trial together. With the extension of public education and ideals of health there is need of an available effective trial case in each school district. The use of such trial lenses will bring to the attention of the public the need for a general correction of ocular defects.

DISCUSSION

DR. W. E. SHAHAN, St. Louis: To get the best results from the trial set, one must not only appreciate its advantages but understand its limitations. The planoconvex lens has a noticeable degree of astigmatism of oblique pencils, though not large, and the patient must still be guarded from looking slantingly through the glasses while the measurements are being made. Also, in the positive forms, especially in the higher powers, the planoconvex lens is virtually weaker than the meniscus lens which the patient will probably wear. If this is not taken into consideration the patient is likely to complain of foggy vision and it will be necessary to add minus spheres to his glasses to bring his vision up to normal.

DR. S. JUDD BEACH, Portland, Me.: The trial frames with small lenses have the advantage of being lighter and also of being much thinner in the higher powers, more like the lenses which the patient will later wear, and of being not so far away from the eye as the larger lenses. They also have certain disadvantages. They are not quite so easy to handle, and one has to be more careful about fixing the axis, as a mistake of a millimeter on the periphery of the smaller lens causes a greater deviation in the axis of the cylinder than happens with a lens of ordinary size.

CLINICAL IMPLICATIONS OF OCULAR DISTURBANCES PRODUCED IN EXPERIMENTAL ANIMALS BY DIETARY CHANGES. DR. ARTHUR M. YUDKIN, New Haven, Conn.

A deficiency of vitamin A in the diet of experimental animals produces ocular lesions known as xerosis conjunctivae, which may progress into xerophthalmia and keratomalacia if the missing factor is not supplied. Difficulty in dark adaptation has been noted in these animals and the clinical picture resembles night blindness observed in man on the same sort of diet. Vitamin A or carotene has been supplied to the

diet of patients afflicted with changes in the choroid and retina. The results obtained are encouraging. Absence of vitamin G, so-called, from the diet of experimental animals produces changes in the lens metabolism so that cataracts are formed. This alteration has been produced in animals immediately after they were weaned. A new field of investigation has thereby been opened. It is possible that a better conception of the formation of so-called congenital cataract is at hand. In an endeavor to produce glaucoma by a diet low in salt I obtained proptosis of the eyeball. The picture resembles that of exophthalmic goiter.

DISCUSSION

DR. C. S. O'BRIEN, Iowa City: Certain symptoms of vitamin A deficiency are similar to certain symptoms of vitamin G deficiency; e. g., the loss of hair about the lids, the edema, the serosanguineous secretion from the conjunctival sac, the matting together of the lids and the corneal changes. The corneal changes are not exactly like those in vitamin G deficiency, in that the corneal surface is dry, oily and covered with fatlike plaques and may ulcerate, whereas, in vitamin G deficiency, most of the pathologic conditions are situated in the layers of the anterior stroma and ulceration does not occur. Dr. P. C. Jeans of the University of Iowa has recently made a study of vitamin A deficiency in children. He believes that night blindness is the earliest subjective symptom of this deficiency. He studied the phenomena of dark adaptation with the photometer. Approximately 10 per cent of the children admitted to the hospital had mild vitamin A deficiency, as shown by retarded adaptation to darkness. In practically all the cases the hemeralopia soon disappeared after the addition of cod liver oil to the diet.

It pleases me to know that the results of the work of Day, Langston and myself on changes in the lens in vitamin G deficiency have been confirmed at Yale. Dr. Yudkin did not mention the corneal changes; they resemble an anteriorly placed interstitial keratitis. Contrary to the experience of the author, we were unable to note any clearing of the lens on feeding vitamin G; however, the process could be stopped at any point by this means. Whether or not the observations on vitamin G deficiency in animals can be interpreted in terms of human lenticular disturbances I am unable to say. It seems reasonable to think that in some instances congenital cataract may be the result of such a deficiency. I have thought that perhaps the changes in the lens in natives of India may be due to some such cause. These people live on starvation rations and cataracts appear in them much earlier in life than they do in the people of the United States.

DR. K. W. COSGROVE, Little Rock, Ark.: It can be taken as proved that animals on a diet deficient in vitamin G will show changes in the lens. In an attempt to show a relationship between deficiency of vitamin G and the lenticular changes in so-called senile cataract, 50 patients have been carefully questioned regarding their intake of foods known to be rich in vitamin G. The patients selected had no known systemic or local cause of cataract, such as trauma or diabetes. The average age was 72. Five foods (milk, greens, meat, liver and yeast) were taken as containing the greater amount of the vitamin G in the diet. A study

of these patients showed that 32, or 64 per cent, were apparently getting very little vitamin G; 17, or 34 per cent, were getting a moderate amount.

Dr. Paul L. Day, Little Rock, Ark.: It might be argued that no human dietaries would ever be free enough from vitamin G to produce cataract. If man's requirement were relatively as low as the rat's that would be true. Until more is known about the human requirement the question cannot be answered. It is known that different species vary widely in the amount of vitamin G necessary to prevent cataract. A very small amount prevents it in the rat, while chicks acquire cataract even with large amounts of the vitamin in their diet. Vitamin G therapy might well be used in cases of incipient cataract. It would do no harm, and it would greatly benefit the patient in other ways.

Dr. Laura A. Lane, Minneapolis: One must be careful in interpreting observations in animals and applying the interpretations to observations in human beings. In many instances, animals do not live under the same conditions or eat the same food as human beings. For example, work has been done on feeding rabbits diets high in fat or high in protein. High blood pressure and vascular changes resulted. The rabbit is not a carnivorous animal; therefore, proper conclusions cannot be drawn. In the same way, while vitamins A and B are important factors there are other elements in the diet, such as the mineral salts; and the body metabolism must be considered. McCollum, Simonds and Becker have shown that not only the lack of vitamin A but a lack of vitamin B and an unfavorable mixture of salts can cause ophthalmia.

Dr. Arthur M. Yudkin, New Haven, Conn.: It is true, as Dr. Lane points out, that many of the ocular changes produced in animals on diets deficient in vitamins have no analogy in man. Nevertheless, the therapy that I propose is not harmful to the patient. I am aware that diet alone may not cure the ocular disease directly, but it is known that a well balanced diet supplemented by the vitamins enriches the body by way of the intestinal tract so that an injured organ can better defend itself. Recently Dr. Adams and her collaborators showed that cataracts were produced by naphthalene much sooner in rabbits fed on a diet of oats and carrots than in rabbits fed on this diet supplemented with cabbage. Cabbage is rich in vitamin G. Is it not possible that the addition of vitamin G delayed the formation of cataracts in the rabbits fed naphthalene?

Standards and Licensure in Ophthalmology. Dr. William H. Crisp, Denver.

The physician is the principal guardian of the public in standardizing medical practice. The public interest is basic. This organization protects the public (1) by directly working for improvement of standards and (2) by increasing the legal requirements. The experience of the American Board for Ophthalmic Examinations ought to be fruitful for improvement in graduate training. Such training should insure that the ophthalmologist goes into his specialty proficient in all branches and particularly those which are of the greatest importance to the greatest number of patients. It may be necessary for the board to

raise its standards and broaden the scope of its examinations. It is doubtful whether the board should continue to issue its diploma on the mere ground of years of practice. It seems logical to presume that the public interest will ultimately require that any one laying claim to qualification for special practice must obtain a special license.

DISCUSSION

DR. T. B. HOLLOWAY, Philadelphia: Time does not permit adequate discussion of Dr. Crisp's criticism of the laws that permit the medical graduate who has passed his examination by the state board to be regarded as adequately qualified to practice medicine or surgery all his life. Many states require in addition to this a hospital internship, and I believe that Pennsylvania was the first to adopt such a requirement.

Ultimately, satisfactory evidence of efficiency will be required before specialization will be recognized or permitted. The important question is: Just how and when is this to be accomplished? I believe that the institutions and the teachers exist, but there is need of money and of students who are willing to accept the training that may be provided. As to the money, few people, even in the medical profession, realize how much it costs to conduct properly a course in graduate education, even when clinical teachers serve without salary, although they may carry a schedule of one hundred and fifty or more hours a year. As to students, there are not many, even today, who desire to take a comprehensive course of two, three or more years' extent.

The increase in the number of opportunities for graduate work as well as in the equipment and teaching in this country during the past twenty years is far greater than is generally recognized and is an important achievement. Short courses of intensive instruction are now available in various parts of the country. A group of well known teachers have willingly given of their time, without compensation, to the carrying on of these courses.

THE FUNCTION OF THE REATTACHED RETINA. DR. PETER KRONFELD, Chicago.

This article was published in full, with the discussion, in the November, 1933, issue of the ARCHIVES, page 646.

INTRACAPSULAR VERSUS EXTRACAPSULAR EXTRACTION OF CATARACT. DR. HARRY S. GRADLE, Chicago.

I have analyzed 200 consecutive extractions of cataract: 100 performed by the Elschnig intracapsular method and 100 by the standard extracapsular method. The results are compared from the standpoint of operative accidents, ultimate vision and postoperative astigmatism.

Statistical Conclusions.—The comparison of the results of 200 consecutive extractions of cataracts, one half by the Elschnig intracapsular method and one half by the extracapsular method or capsulotomy, shows but few differences except in secondary needlings and in vitreous losses. Here, the percentages are markedly in favor of the intracapsular method. In the matter of prolapse of the iris, postoperative astigmatism and final vision there is little to choose between the two methods.

Impression Conclusions.—From the work performed in this series of cases I have gained some definite impressions which cannot be corroborated statistically, but which, to my mind, are positive, as follows: 1. The ideal operation for removal of cataract today is the Elschnig intracapsular extraction with a round pupil. That operation, however, is not for the casual operator; it requires the nicety of feel that can result only from continuous operating. 2. If an attempt to remove the lens within the capsule is not successful removal can be completed as an extracapsular extraction without any added dangers. 3. After intracapsular extraction there is less irritation of the eye than after extracapsular extraction. 4. The healing process is more rapid after intracapsular extraction. 5. Following extraction by the Elschnig method there is not the distortion of the pupil that occurs so frequently after other types of intracapsular extraction. 6. In the hands of an experienced operator, the Elschnig method of intracapsular extraction is just as safe, if not more so, than the older method of capsulotomy.

DISCUSSION

DR. ARNOLD KNAPP, New York: In describing the technic of the intracapsular operation Dr. Gradle stated that he applies pressure only after dislocation has taken place. In so doing, he loses a valuable adjunct in the subluxation of the cataract. Manipulation with the capsule forceps and pressure on the eyeball at the lower limbus should be practiced at the same time. Dr. Gradle did not emphasize the importance of the step of getting the cataract to tumble, which is the greatest safeguard against loss of vitreous.

The results of intracapsular delivery in Dr. Gradle's series are of interest: he stated that in 20 per cent the capsule ruptured during extraction and had to be extracted separately. This is a very high percentage; it proves to my mind the defect of the traction method of intracapsular extraction, which I have repeatedly tried to point out. If, on the other hand, the forceps is released as soon as the cataract is subluxated, and pressure is then applied, as in the Smith technic, fewer capsules will be ruptured in the extraction of the cataract.

The escape of vitreous is interesting. This complication occurred in 9 of the extracapsular and in 3 of the intracapsular series. This is very unusual, as even the most enthusiastic intracapsular operator must acknowledge that the percentage of the losses of vitreous is greater with the intracapsular procedure. Dr. Gradle did not state whether the losses of vitreous occurred before or after the extractions of cataract.

Unfortunately, he did not give in detail any differences in the two types of operation, that is, in the healing or in the incidence of iritis or other complications (glaucoma and retinal detachment), which are so important in judging the value of an operative method.

DR. WALTER R. PARKER, Detroit: I agree with Dr. Knapp that no one method of procedure is applicable for all cases requiring extraction of a cataract, and I believe that the more successfully one selects cases for the various methods of extraction, the better will be the results.

DR. WARREN D. HORNER, San Francisco: The intracapsular operator must possess unusual dexterity, a satisfactory technic acquired through working with a master operator, judgment as to which cases

he should or should not attempt to treat by this method and, lastly, proper assistance. To acquire and maintain operative dexterity, one must have adequate clinical opportunities, usually in addition to private practice. For many of us, circumstances will not allow the fulfilment of all of these requisites. Akinesia, deep orbital injection, suture of the superior rectus muscle, suture of the conjunctival flaps and proper preoperative medical preparation of the patient are available for all, regardless of the type of operation. These have done much to lessen unforeseen hazards originating in the patient himself, through the elimination of pain and apprehension. At the University of California, sutures of the lid have been substituted for any type of metallic control of the lid. These sutures are shaped like the letter "W." They have proved most satisfactory during the past three years and are safe though employed by an untrained assistant.

Dr. Jasper M. Molsberry, Iowa City: I wish to present a series of 282 cases of attempted intracapsular extraction of cataract performed in Dr. O'Brien's clinic at the University of Iowa. Of the intracapsular operations, 157, or 56 per cent, were successful. Dr. O'Brien performed approximately 50 per cent of the operations; the remainder were performed by the men in the service and included their first attempts at extraction of a cataract. When the capsule ruptured, the extraction was completed by the extracapsular method. Complete iridectomy was performed in 139 cases, peripheral iridotomy in 89 cases and simple extraction in 54 cases. It is interesting to note that among the simple extractions there were 8 prolapses, or 14.8 per cent; among the peripheral iridotomies there were no prolapses and 1 incarceration; among the complete iridectomies there were 4 prolapses, or 3 per cent. There was loss of vitreous in 21 cases, and there were needlings in 5 cases.

Dr. Otis R. Wolfe, Marshalltown, Ia: Among 800 cataract extractions, 3 were followed by hypertension and loss of vision. In all 3 the capsule and débris from the lens were retained. Two of the 3 were in patients with a history of glaucoma, and a tentative diagnosis had previously been made. The Barraquer technic ruptures fewer capsules than any of the technics in which forceps are used and therefore, it is the operation of choice, especially in the extraction of immature and hypermature cataracts.

Dr. Walter B. Lancaster, Boston: With traction and pressure, one is apt to pull a little too hard and tear the capsule; also, if one uses pressure with traction, one is more apt to observe loss of vitreous and other complications. Elschnig definitely stated that he grasps the capsule and for about six or eight seconds moves it laterally to and fro to dislocate the lens, and then expresses. Šafář, who visited Elschnig, went back to Vienna and tried to apply this method, but with poor results; he then modified Elschnig's method and obtained good results. Šafář's method is something like this: He grasps the lens with a Kalt forceps and simultaneously exerts pressure on the outside and traction on the inside, dislocating the lens by the combined effect of the pressure and traction. He then continues to exert combined traction and pressure in getting the lens out, either with or without tumbling. I do not dilate the pupil before the patient comes to the operating room but dilate by the combined effect of cocaine and epineph-

rine. If the pupil cannot be dilated the case is not suitable for extraction by this method through the intact pupil. If it can be dilated satisfactorily, the lens is extracted by combined pressure and traction as described. The iris is then replaced and a peripheral buttonhole iridectomy done according to the original method of my old chief, Dr. Chandler. All cases must be considered in regard to the age of the patient. Young patients are not suitable for extraction of the lens in the capsule. Patients with prominent eyes should be handled with great circumspection. Particularly dangerous are patients with very fat heads; their eyes are under more or less pressure from the fat in the orbit.

Dr. Vernon A. Chapman, Milwaukee: Dr. Gradle did not mention the Barraquer method, which is the only one of pure extraction, the rest employing combined expression and extraction, or expression alone. I mention this for the reason that I had the opportunity to try the Barraquer method in a variety of cases and to determine whether it could be used in all types of cases. It can be, but it is better for some cases than for others.

Dr. Harry S. Gradle, Chicago: Possibly Dr. Knapp misunderstood the purpose of my paper, which was not to discuss the various methods of intracapsular operation but merely to compare the results that I obtained by using one method of intracapsular operation with the results that I obtained in a similar number of cases by using extracapsular operation. The statistics are only personal. They were not compared with those reported by other men, and I offered them to you simply because I thought you might be interested in hearing what the end-results were in using the extracapsular method and one type of intracapsular method, the same person operating under almost identical circumstances.

Complications of Extraction of Cataract in Relation to Intra-Ocular Tension and Blood Pressure. Dr. Oscar B. Nugent, Chicago.

This paper reports a study of complications during or following extraction of cataract in relation to intra-ocular tension and blood pressure. An analysis made from tabulations tends to show that the intra-ocular tension is a more dependable sign than the blood pressure as a guide in anticipating complications in this operation. A brief review of some important extracts from the literature is given. Intra-ocular tension and blood pressure are found to be lower in the Indian than in the American patient.

DISCUSSION

Dr. John Green, St. Louis: The patient with very high vascular tension is not a good subject for extraction of cataract. Should the surgeon be so fortunate as to escape the major catastrophe—expulsive hemorrhage—there is always the possibility that the sudden reduction of intra-ocular pressure to zero may induce small hemorrhages in some of the retinal vessels. Should this not occur, the visual result may be far from satisfactory on account of the presence of angiosclerosis of the retinal vessels, with attendant hemorrhages and exudates. The presence

of high ocular tension calls for special precautions, for in such a case one is dealing with a glaucomatous eye in which the cataract is incidental. If the tension is at or above the upper limits of normal, reduction may be brought about by the use of miotics. When there is a considerable increase in intra-ocular tension, preliminary iridectomy should always be performed, followed by extraction a few weeks later. In such cases atropine is absolutely contraindicated, both before the extraction and following it.

SOME POSSIBILITIES OF ORTHOPTIC TRAINING. DR. GEORGE P. GUIBOR, Chicago.

This article appeared in full, with the discussion, in the March, 1934, issue of the ARCHIVES, page 433.

THE ARTERIOLES OF THE RETINA IN TOXEMIA OF PREGNANCY. DR. HENRY P. WAGENER, Rochester, Minn.

Spastic lesions of the arterioles are the most frequent and usually the primary signs of retinal involvement in toxemia of pregnancy. Such lesions occur both in acute toxemia and in toxemia superimposed on previous vascular or renal disease. They occur in about 70 per cent of cases of toxemia. In about 60 per cent of the cases of spastic lesions, the lesions disappear with the termination of pregnancy, and the blood pressure returns to normal or to its previous level. In about 40 per cent of the cases, organic lesions develop in the arterioles, often in association with retinitis. In such cases the elevation of blood pressure usually persists. Diffuse retinitis of the albuminuric type is to be regarded as evidence of severe generalized arteriolosclerosis rather than of primary nephritis. Primary detachment of the retina occurring in the course of acute toxemia does not have the same serious clinical significance as diffuse retinitis.

DISCUSSION

DR. ROBERT J. MASTERS, Indianapolis: The appearance of the picture called "pregnancy retinitis" seems to follow no rule, as the most severely toxic patients may have no retinal exudates or hemorrhages. Constriction of the retinal arterioles is constantly seen, however, in all patients whose blood pressure is elevated to 150 or more systolic and 100 or more diastolic. For the purpose of proving this arterial constriction by measurement I have employed an ophthalmoscope that projects a linear graticule on the eyeground. Changes in the retinal arteries probably indicate, to a reasonable extent, changes in the arterioles of the rest of the body. Constriction of the retinal arteries therefore suggests a generalized arteriolar constriction sufficient to elevate the blood pressure. This arteriolar constriction is probably the result of irritation produced by a toxin circulating in the blood stream. If the toxic irritant is allowed to continue its action too long before pregnancy is terminated, its effect will advance from the stage of irritation with arteriolar constriction to a stage of destruction with sclerotic changes in the vessels, permanent vascular hypertension and chronic nephritis of various degrees of severity.

Dr. Arthur J. Bedell, Albany, N. Y.: As I understand Dr. Wagener, his plea is that when an ophthalmologist is called on to render an opinion on conditions found in the fundus of a pregnant woman suffering from toxemia, he should urge and insist on the removal of the cause of the toxemia during the stage of arteriolar spasm, instead of waiting until the lesion in the vessel becomes fixed.

Ectopia Lentis, with Report of a Case of Total Dislocation Directly Downward. Dr. Warren D. Horner and Dr. Sol Maisler, San Francisco.

A case of congenital bilateral total dislocation of the lens is reported in a boy of 10, the dislocation being directly downward. Such a position is stated by most authors to be exceedingly rare, while some deny its occurrence. It was accompanied in this case by an old monocular chorioretinitis and a persistent partial pupillary membrane but not by any other congenital anomaly and not by glaucoma. In order that the aphacia might be overcome, bifocal lenses were prescribed and have been successfully worn for the past year, with definite improvement in both distant and near vision.

DISCUSSION

Dr. William Zentmayer, Philadelphia: The outstanding feature of this anomaly is its hereditary and familial tendency. Instances even more striking than those noted by these authors have been reported. R. M. Gunn recorded its occurrence in 17 members of 5 families with 22 children. Dehenne and Bailliart reported a history of 4 generations affected in which all the 6 descendants of the first showed double luxation. There are a number of similar histories recorded in the literature. The descent seems not to be of like nature in all cases. Gunn stated that in the case he observed it was not a mendelian recessive. In 2 families it acted as a pure dominant. In the case described by Franceschetti, it was a recessive hereditary condition manifested through consanguinity, while Kotlarveskaid considered it to be of a hereditary dominant type, basing his opinion on observations in 13 cases. Whatever the genesis of the condition may be, two factors are significant: the occurrence at times of a coincident corectopia and the frequent association of myopia (in 60 per cent). The first indicates the persistence of a mesodermic strand as the cause of both anomalies, and the latter, a stretching of an inherited weak zonule, as the more the eye stretches the more the fibers are stretched. This probably accounts for the fact that the ectopia increases with time and in many instances becomes a complete luxation (Rötth). The myopia is not in every case axial but, as stated by the authors, is sometimes lenticular. In one of the cases that I observed, the fundus presented myopic changes. Butler, Meesmann and Hegner reported instances of downward dislocation. In a number of cases there was a deformity of the skull, in some cases associated with impaired mentality. Operation, when called for by secondary glaucoma or because of cloudiness of the lens, presents difficulties. The statement that needling entails transfixation is a correct generalization, but in individual cases needling, as ordinarily done, has been successful.

MIXED (TERATOID) TUMORS OF THE LACRIMAL CARUNCLE. DR. DERRICK T. VAIL, JR., Cincinnati.

This article was published in full, with the discussion, in the November, 1933, issue of the ARCHIVES, page 593.

STUDIES OF THE VISUAL FIELDS IN PATIENTS WITH FUNCTIONAL HEADACHES OF PITUITARY ORIGIN. DR. BEULAH CUSHMAN, Chicago.

From a careful study of 10 patients (9 females and 1 male) I have arrived at the following conclusions: 1. Certain periodic attacks of headaches and discomfort of the eyes simulating those accompanying ciliary spasm may be due to dysfunction of an organically sound pituitary gland or to distention of its capsule by physiologic hypertrophy. 2. The majority of the visual fields in such cases show a contraction of the superior temporal quadrant for color and varying changes in the form fields. 3. The aid of the internist and that of the roentgenologist should be sought in a study of the activity of the pituitary gland and the condition of the sella.

DISCUSSION

DR. CLIFFORD B. WALKER, Los Angeles: Dr. de Schweinitz showed, in the Bowman lecture of 1923, that the chiasm is not always in contact with some part of the sellar diaphragm. Indeed, in many cases, the gland would have to double in size before touching the chiasm, and even then the chiasm has some chance to move away because it is suspended or stretched in subarachnoid mesh and fluid. Horizontal variations, as cited by Dr. Cushman, add to this vertical variation. The chiasm has no dural covering, however, as does the hypophysis, and without this protection is more quickly affected by pressure. Nevertheless, it seems clear that, of a given number of persons having swelling of the hypophysis sufficient to produce pain from its dural capsule and diaphragm, only a certain number, perhaps no more than 50 per cent, may be expected to have a defect in the upper part of the temporal field from pressure. On the other hand, I know from examination of many patients with hypophyseal struma or tumor that of those having sufficient swelling to give a marked defect of the field the percentage having headache is not greater than 50. Evidently, much depends on the sensorium of the individual patient and particularly on that of the dura of the diaphragm and the neighborhood. Apparently, it is necessary to fall back on the possibilities of circulatory disturbance and secondary toxic manifestations to explain cases with no chiasmal pressure, yet with defects of the visual fields. In connection with the search for defects of the visual fields in a patient having headache of the pituitary type I wish to emphasize the value of quantitative perimetry.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS: A LAY MOVEMENT FOR THE CONSERVATION OF VISION. DR. WILLIAM CAMPBELL POSEY, Radnor, Pa., and LEWIS H. CARRIS, New York.

This article was published in full, with the discussion, in the November, 1933, issue of the ARCHIVES, page 621.

CONJUNCTIVAL VESSELS. DR. A. D. RUEDEMANN, Cleveland.

The vascular system of the conjunctiva lies in two planes: one, that of the superficial layer, and the other, that of the branches of the anterior ciliary arteries. There is a heavy capillary plexus at the limbus.

The speed of the blood flow, the size of the vessels and the action of various substances on the vessels can be studied easily in the conjunctival vascular system. This field apparently offers an excellent location for the physiologic study of blood vessels, as all action is in living tissue in normal position and under controlled conditions.

Pathologically, minute hemorrhages are commonly found. Aneurysmal dilatations are more common than in the retina. Thrombi and emboli apparently are present more commonly than has been suspected.

It seems likely that when normal standards have been established, the study will reveal sufficient information to warrant investigation of the conjunctival capillaries in all cases in which disease of the blood or of the vessels is suspected.

The vessels can be measured and counted and, to date, the patients have experienced no deleterious effect from the illumination.

THE O'CONNOR CINCH SHORTENING OPERATION FOR HETEROTROPIA AND HETEROPHORIA. DR. GEORGE N. HOSFORD and DR. AVERY M. HICKS, San Francisco.

This article appears in full in this issue of the ARCHIVES, page 814.

POCKET-FLAP SCLERECTO-IRIDODIALYSIS IN GLAUCOMA. DR. CHARLES NELSON SPRATT, Minneapolis.

I now have records of 28 eyes on which sclerecto-iridodialysis has been performed.

The cause of primary glaucoma is not known. The relief of the symptoms due to pressure by the operation of decompression (sclerectomy) and the production of a filtrating cicatrix is based on sound surgical principles.

The pocket-flap of conjunctiva gives a sterile field for the operation, lessens the danger of immediate and late infection and covers the opening in the sclera with firm protective tissue.

Iridodialysis removes the block of the angle of the anterior chamber better than iridectomy. No iris remains at the base to cause recurrence. There is no dazzle or glare after the operation, for the pupil is small, round and central, and, as the sphincter is not injured, miotics may be used after the operation.

DISCUSSION

DR. WALTER B. LANCASTER, Boston: My feeling has been that an operation on the iris which permanently liberates the angle for 5 mm. or more would relieve the patient in probably 60 per cent of cases of glaucoma. If it is unsuccessful, a filtration operation can be performed.

Dr. Spratt invites comparison with iridencleisis. The important basal fact concerning iridencleisis is that the presence of iris in the scleral incision greatly favors filtration. However, it makes a great deal of difference how the iris is placed in the scleral incision. The pigment epithelium is the important element; it must not be folded on itself or lacerated.

Dr. Virgil J. Schwartz, Minneapolis: The fact that so many operative procedures have been proposed for the relief of glaucoma, not only varying in manner of execution but diametrically opposed in basic principle, is sufficient evidence that not one of them has thus far been found to be uniformly satisfactory. Moreover, it is possible that until the fundamental cause of glaucoma in its various forms is discovered there will be no unanimity on this subject. Particularly is this true of so-called chronic simple glaucoma. Dr. Spratt employs two of the more generally accepted operations together, the Lagrange sclerectomy and iridodialysis. It might be argued that if either of these is entirely successful it should not be necessary to do both. The fact remains, however, that the reduction of tension produced by iridodialysis may not always be permanent, so that removal of a piece of sclera is advisable. Dr. Spratt fortifies his result by using the pocket-flap, which he has been employing in operations for cataract for several years. Moreover, by tearing the iris instead of cutting it, he is fairly certain not to leave a margin of iris tissue at the base, as so often happens with iridectomy.

Spontaneous Dislocation of the Lacrimal Gland. Dr. James W. Smith, New York.

The literature on spontaneous dislocation of the lacrimal gland is reviewed. The diagnostic signs and etiologic factors are considered. A case of bilateral dislocation of the lacrimal gland is reported, and a technic for replacement of a dislocated gland is presented wherein the gland is restored to its normal anatomic situation without sacrificing any part and at the same time the relaxed tarso-orbital fascia is utilized as a curtain to keep the gland in place. In the case reported a satisfactory and permanent result was obtained.

Spontaneous dislocation of the lacrimal gland is a separate disease entity, seldom recognized by American ophthalmologists and often erroneously considered to be a complication of blepharochalasis and occasionally of ptosis adiposa. The disease is seen commonly in Russian patients and is probably of more frequent occurrence in this country than is indicated in American textbooks and literature.

Extirpation of a dislocated lacrimal gland is a hazardous operation which should be replaced by the safer and simpler procedure of returning the gland to the lacrimal fossa.

If the latter operation is performed early in life, it will prevent the formation of the overhanging folds of skin in the upper lids.

DISCUSSION

Dr. F. Herbert Haessler, Milwaukee: Dr. Smith assumes that in the cases he has observed the seat of the lesion was probably the palpebral orbital fascia, which allowed the gland to prolapse, with secondary changes in the gland and in the skin. This is somewhat comparable to what happens in inguinal hernia. There also are some holding structures, which finally give way under pressure from within. Dr. Smith's operation seems to be successful; at least the pictures show that he accomplished what he attempted. He assures me that this operation is not very difficult. The fact that extirpation of the tear gland is a

dangerous procedure has not been sufficiently emphasized in the literature.

Dr. Edward Jackson, Denver: The term applied to the condition is often of doubtful propriety. The dislocations that I have seen have all been mislocations or anomalies of location of the gland. The orbital gland was not in the orbit but partly in the lid, entirely independent of enlargement. The condition was a developmental anomaly. In none of the cases were there any symptoms that caused the patient to seek relief. The condition was noticed when the eyes were examined for other reasons. It has never occurred to me to do anything for any of these patients. I have certainly seen 5, and probably more, cases that should be put in this class.

Dr. Vernon A. Chapman, Milwaukee: Confusion is caused by the use of the term "lacrimal fossa." Unfortunately, there are two separate fossae to each of which the term "lacrimal fossa" is applied. These are the fossa glandulae lacrimalis and the fossa sacci lacrimalis—the fossa of the lacrimal gland and the fossa of the lacrimal sac—in two obviously quite different anatomic locations. As the lacrimal gland does not begin secretion of tears before the second to seventh week after birth, and the infant's eyes get along very well without such secretion, there is reason to believe that compensation for its loss later in life may be established without great danger to the eyes. If an operation for replacement is to be done, prolapse of the lacrimal gland should be differentiated particularly from dacryops or cyst of the lacrimal gland and from tumors, which may range in size from that of a pea to that of a pigeon's egg and are usually not inflammatory.

Dr. James W. Smith, New York: I was interested in Dr. Jackson's statement that he had seen 2 patients in the same family who exhibited a tendency toward dislocation of the lacrimal gland. The prominent orbital roofs evident in my patient were also present in his father and his paternal aunt, both of whom were operated on by me for glaucoma. I was informed that prominent orbital roofs were also present in the patient's paternal grandfather. Many cases of dislocation of the lacrimal gland are discovered accidentally in routine clinical examinations of patients who come for treatment of other ophthalmic conditions. My patient's chief complaint was of repeated attacks of dacryo-adenitis of the left side rather than of the cosmetic deformity produced by the overhanging folds of skin in the eyelids.

PHILADELPHIA COUNTY MEDICAL SOCIETY, THE EYE SECTION

Nov. 14, 1933

Charles R. Heed, M.D., *Chairman*

Sidney L. Olsho, M.D., *Secretary*

A Case of Retinitis Striata. Dr. M. R. Gabrio.

The patient, a young man, stated that he had attacks of epilepsy. An opinion was expressed that a traumatic detachment of the retina preceded the development of retinitis striata.

TWO UNUSUAL CASES OF TUMOR OF THE FUNDUS. DR. CHARLES W. LEFEVER.

In discussing one instance, Dr. LeFever expressed the belief that the patient did not have a solid tumor but a fluid retinal detachment. The second case, however, was regarded as one of sarcoma.

BILATERAL PANOPHTHALMITIS IN A CHILD. DR. FRANK A. MURPHY.

This case occurred during the course of an acute cerebrospinal meningitis. Those who saw the patient and discussed the case suggested that the term "exudative choroiditis" or "pseudoglioma" would be better than "panophthalmitis."

TWO CASES OF DERMOID CYST OF THE ORBIT FROM THE CLINIC OF DR. LOUIS LEHRFELD, PHILADELPHIA GENERAL HOSPITAL. DR. E. J. DONNELLY.

In discussing these cases, Dr. Lehrfeld explained the ease of extirpation in one instance and the difficulty involved in the second because of the depth of the cyst in the orbit. Microscopic sections of the excised cysts were demonstrated.

A CASE OF CONGENITAL SUBLUXATION OF THE LENS AND IRIDODONESIS. DR. MARY BUCHANAN.

A CASE OF SARCOMA OF THE LEFT MAXILLARY ANTRUM PRODUCING PROPTOSIS AND CHOKED DISK. DR. LITTLE.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Nov. 10, 1933

A. C. HUDSON, F.R.C.S., *President*

CONGENITAL ABNORMALITIES OF THE IRIS AND LENS. MISS IDA MANN.

In 1924 I showed some embryologic specimens bearing on the mechanism of the production of coloboma iridis. At a certain stage in normal embryos of human beings and of other mammals the capsulopupillary vessels as they curl over the rim of the optic cup produce small indentations in this rim. One of the vessels in an abnormal human embryo had persisted in the production of an indentation beyond the usual stage and seemed to be about to cause a coloboma. If this is a common factor in the genesis of coloboma, it should be possible to demonstrate clinically some trace of the abnormal vessel. Occasionally its presence can be demonstrated in adult life. In 1900, von Hippel showed a section of an adult eye with a large vessel pulling the iris backward at the apex of a coloboma.

The following points were brought out in 6 cases that I have studied:
1. Persistence of a vessel can occur in any situation round the equator

of the lens. 2. There is always a gap in either the ectodermal or the mesodermal portion (or both) of the iris. 3. Evidences of persistent pupillary membrane are also found, either as tags from the lesser circle or as spots of pigment on the anterior portion of the capsule of the lens. 4. If the zonule can be seen, a gap is observed in it. 5. Usually there is a notch in the margin of the lens. 6. Often there is an opacity in the lens, localized in the region of the coloboma. 7. The persisting vessel can sometimes be seen, but more often it has atrophied after producing the abnormalities.

Metastatic Carcinoma of the Choroid. Mr. A. C. Hudson.

Mr. Usher reported 110 cases of tumor of the choroid in 22 of which the primary growth was elsewhere than in the breast. In a total of 149 recorded cases of carcinoma of the choroid there were only 51 in which the primary growth was in a part of the body other than the breast. In many of the reports the tumor of the choroid was described as an adenocarcinoma.

A man, aged 41, was admitted to the Royal London Ophthalmic Hospital in August, 1910, with a history of blurred vision in the right eye for eleven days. There had also been localized pain in the lumbar region for a month. A large, round, yellow swelling of the choroid extended up and out from the disk, and the retina was wrinkled on the surface. The eye was excised the next day. Seven months later the patient died with signs of malignant disease involving the thorax, the right lung and the vertebral column. The tumor of the choroid was a papilliferous adenocarcinoma. Refusal to permit postmortem examination prevented the determination with certainty of the site of the primary tumor.

An Optometer. Mr. J. D. Magor Cardell.

The optometer is an instrument for testing the sight of bedridden patients or of those who have to be seen in their homes. It weighs only 8 ounces (226.8 Gm.). It is possible by means of this instrument to carry out the Snellen distance test and the duochrome test, and the use of the rotating fan for the detection of astigmatism is also practicable. Any conveniently placed light is viewed through the tube, which is held in contact with the cheek of the patient. The cap at the other end may be exchanged for any of the other testing devices mentioned. There is enough room between the tube and the eye to accommodate a trial frame and to allow the use of a crossed cylinder.

Kayser-Fleischer Ring in the Cornea in Wilson's Disease. Dr. S. A. Kinnier Wilson.

Lenticular degeneration is very rare, and the Kayser-Fleischer ring is even more rare. I have seen this type of ring in 3 cases, including one observed in Denmark. The boy and the girl presented came to the outpatient department of King's College Hospital with a tentative diagnosis of the so-called postencephalitis. The corneal ring seems to consist of a deposition of minute granules of pigment on the under surface of the cornea and can be seen through the cornea. Both children

have mild cirrhosis of the liver, though the levulose tolerance tests were negative.

ARACHNODACTYLY WITH OCULAR SIGNS. MR. E. F. KING.

The first patient, a woman, aged 37, has defective vision in both eyes, which glasses have not improved. On examination, the right eye showed iridodonesis, and the pupil dilated moderately in response to mydriatics. There was ectopia lentis up and in, and a diffuse haze was noted throughout the lens, with fine vitreous opacities; there was scarring of the choroid below the disk. Vision was 6/60; with a plus 1 diopter sphere, it was 6/36.

The second patient, aged 18, is a daughter of the first patient. Her vision has never been good. Iridodonesis is present in both eyes. She is of normal build but has unusually long fingers.

The third patient, aged 13, is also a daughter of the first patient. Her left eye is the more defective of the two. She has always worn glasses. She has a coloboma lentis medially; otherwise her condition is much like that of her sister.

DISCUSSION

Mr. Hudson suggested the possibility of an iridotomy of the lower part of the sphincter and remarked that patients with real arachnodactyly usually have a short life.

CONGENITAL NUCLEAR HYPOPLASIA. MR. FRANK W. LAW.

In the first patient, the condition has been present since birth, and there is bilateral facial palsy. The movements of the eyes are limited to depression and elevation. There is defective closure of the lids; when closure is attempted the eyes roll under the upper lid.

The second patient is a girl with bilateral facial palsy and convergent squint. She has limited adduction in each eye. She is receiving electrical treatment for her facial condition, and improvement has commenced.

The third patient, a boy, has deficient elevation and no abduction, and convergence is either very weak or absent.

These 3 cases support the oft-expressed view that other congenital deformities occur in association with the ocular condition, as the first patient had clubfoot, which responded to fifteen operations, the second has harelip and the third had a nevus and has a rudimentary accessory auricle and an undescended testicle.

TUBERCULOUS KERATITIS TREATED BY LOCAL ULTRAVIOLET IRRADIATION. MR. FRANK LAW.

This patient had been treated with the usual remedies for tuberculous keratitis, and as there was little response, it was decided to try the effect of ultraviolet rays.

When I first saw her the condition of the left eye was worse than that of the right. The left eye has received a total of three hundred and forty minutes of irradiation, and the right eye, one hundred and thirty-five minutes. The dosage was five minutes three times weekly.

The treatment has helped both eyes, the left more than the right.

MONOCULAR PROPTOSIS (LEFT) WITH RETINAL CHANGES. MR. MONTAGUE L. HINE.

This woman, aged 44, was first seen at the hospital in May, 1932. She at that time said that her left eye had been prominent for a few weeks. The sight, she said, was blurred. On examination, the vision of the right eye was found to be 6/6; that of the left was limited to perception of fingers at a distance of 0.5 meter. The pupils were active and equal. The left eyeball was proptosed directly forward. When the globe was pressed backward there was some tenderness, but movements in all directions were good. There was a large temporal paracentral scotoma to 1 degree, and three deep retinal bands extended down and out from the optic disk. The Wassermann test was negative. The urine was normal. There was no sign of exophthalmic goiter or of anything abnormal in the central nervous system.

CYSTS OF THE CONJUNCTIVA. MR. RYCROFT.

The condition presented by this patient is bilateral. It seems to have originated in the palpebral conjunctiva and to have spread backward. At operation, I shall aspirate the cyst and inject dye, in order to see if there are any extensions that cannot be detected by the naked eye.

PSEUDOGLIOMA. MR. A. C. HUDSON.

The chief point in favor of a diagnosis of pseudoglioma in this case is evidence of former inflammation of the iris. There are many fine strands of adhesions, and a small amount of gray membrane can be seen in the center of the pupil. The anterior chamber is more shallow on the left side than on the right. If the condition is true glioma, the eye should be removed.

Dec. 8, 1933

A. C. HUDSON, F.R.C.S., *President*

OCULAR TUMOR. MR. LINDSAY REA.

A woman, aged 77, showed a small spot on the iris which had been diagnosed as a malignant growth. On local advice, radium was applied, but the only result was a stubborn conjunctivitis. No iritis was visible with the slit lamp, but in the gray iris a brown area suggested sarcoma of the iris, already beginning to spread. The eye was removed, and the section showed a spindle cell sarcoma. The growth did not reach the ciliary body.

A man, 64 years of age, who had been tuberculous since birth, presented a small fatty tumor above the right eye, lying on the insertion of the superior rectus muscle. Aspiration indicated that the tumor was solid. Under cocaine anesthesia the tumor was shelled out easily after peeling back the conjunctiva. In the pathologic report a few days after the operation it was declared to be round cell sarcoma, and later it was proved to be true lymphosarcoma, a metastasis from

a lymphosarcoma elsewhere. At a subsequent examination, the eye was seen to be completely enveloped by a recurring growth which had pushed the eye outside the orbit. The tumor was not adherent to the periosteum and was easily shelled out, with the contained eye, under procaine hydrochloride anesthesia. Recovery was uneventful. The spleen at that time extended half-way across the abdomen and was evidently the site of origin of the tumor.

MALIGNANT MELANOMAS OF THE CONJUNCTIVA AND EYELIDS. MR. A. RUGG-GUNN.

A man, aged 64, was first seen five years ago. In the palpebral and bulbar conjunctiva of the right eye he showed many irregular, deeply pigmented patches which had been present since birth. A year later the patches did not seem to have changed. Three years later a black, pedunculated growth, 4 mm. in diameter, was seen attached to the bulbar conjunctiva, near the semilunar fold. It was excised and reported to be melanotic sarcoma.

In another patient melanoma involved the palpebral skin and conjunctiva. On attempted excision of the nodule from the lid, it was found to permeate the whole thickness of the lid. The orbit was then exenterated, and section showed the lesion to be sarcomatous.

These growths were radiosensitive, but there appeared to be a difference in radiosensitivity between those of the palpebral and those of the bulbar conjunctiva. The multiplicity of the tumors constituted a problem, since a lethal dose to one might be a stimulating dose to the others.

DISCUSSION

MR. HUMPHRY NEAME: If the condition is sarcoma, I think there is a fair chance that radium will destroy the growths, but there is a risk of causing cataract. It would be uncomfortable to have needles buried under the conjunctiva and retained there for days.

COLOBOMA OF THE IRIS AND CHOROID. MR. A. HUGH THOMPSON.

Of eight cases, three were observed in males and five in females. In four, nystagmus was present; in four, it was absent. Three of the eight patients had microphthalmos in one or in both eyes. Myopia was found in ten of thirteen eyes. Of fourteen eyes tested, nine had various degrees of blindness, from 3/60 down. In three eyes the vision was 6/60; in two, 6/36.

DIATHERMIC MULTIPUNCTURE. MR. J. COLE MARSHALL.

A woman, aged 72, with sudden failure of sight, was found to have a projecting detachment of the retina in the upper temporal quadrant. There was a horseshoe tear. Scleral puncture was done 14 mm. from the limbus, and considerable fluid resembling vitreous humor escaped. Four days later the retina was reattached, though a hole was not visible. Two days later the retina was again detached; a hole was now visible, and a day or two later the detachment was complete. Multiple scleral punctures were therefore employed, and the retina is now back in place. With correction, the vision is 6/24.

ŠAFÁŘ'S METHOD OF DIATHERMY. MR. COLE MARSHALL.

Two years ago a man, aged 59, who had not suffered any trauma felt that an air bubble was floating in one corner of his eye. He was found to have retinal detachment in the upper temporal quadrant, and toward the periphery, dots of pigmentation. Two tears could be seen. Diathermic multipuncture by Šafář's method has resulted in reattachment, and the man is again at work as a tailor.

Mr. G. Penman also showed a case of retinal detachment treated by Šafář's method, resulting in complete restoration with the full field of vision.

TRAUMATIC MONOCULAR ARGYLL ROBERTSON PUPIL. MR. RUGG-GUNN.

A boy, aged 9, was knocked down by a car about seven months prior to presentation and was delirious for some days. Roentgenograms showed a fracture of the vault of the skull and rupture of both tympanic membranes. After the accident all the movements of the right eye were absent, but ptosis was not complete. There was no proptosis. Vision was not affected, but there was loss of the power of upward movement of the right eye and of its accommodative power. It is the general opinion of colleagues at the hospital that the abnormal pupil is caused by hemorrhage in the orbit affecting the ciliary ganglion.

THROMBOSIS OF THE CILIORETINAL ARTERY. MR. EUGENE WOLFF.

In a woman, aged 68, the vision in the right eye failed to the level of 6/60, and the right pupil reacted moderately. Three weeks later, toward the outer side of the disk there was a pale, edematous area fringed with hemorrhage, and one could see the break in the cilioretinal vessel. Five days later the pale, edematous area became converted into the dense white of coagulation necrosis. On December 2, the vision in the right eye was less than 6/60, and the pupil was scarcely active.

UNILATERAL PROPTOSIS. MR. A. C. HUDSON.

A boy presents a mass in the orbit, located above and behind the eye, producing proptosis. Chronic inflammatory conditions can be excluded, and the clinical evidence suggests cavernous angioma.

DISCUSSION

MR. HUMPHRY NEAME: Has a primary suprarenal tumor affecting the medulla been considered?

MR. A. C. HUDSON: The boy has been carefully examined for abdominal disease, without result. Hypernephroma usually affects both orbits, and if the growth were malignant, one would expect more growth during the year.

Book Reviews

Experimentelle Studien über den Verlauf der Tuberkulose im Kaninchenauge. By Dr. Ilona Krasso, I. Universitäts-Augenklinik, Vienna. Pp. 138, with 35 illustrations. Berlin: S. Karger, 1933.

The author performed experiments on the eyes of twenty-seven rabbits to study the effect of injury on tuberculous infection. The experiments were divided into six groups: (1) infection plus injury, (2) infection plus injury plus from one to three subconjunctival injections of a tuberculin preparation, (3) infection plus the introduction of sterile copper, (4) infection plus superinfection, (5) the introduction of copper plus superinfection and (6) infection with tubercle bacilli from patients suffering from polyarthritis and erythema nodosum, plus injury.

In every case the infection was produced by the introduction of a culture of tubercle bacilli into the vein of an ear. In most animals the tubercle bacilli were from a patient with severe chronic uveitis in the clinic of Prof. J. Meller, whose case was reported by him in the *Zeitschrift für Augenheilkunde* (**77**: 1 1932). The injury consisted of a cut in the upper part of the rabbit's eye, 4 mm. long, parallel with or radial to the limbus, followed by prolapse of the vitreous and, in most instances, of the ciliary body. The injury was produced from twenty-one to twenty-six days after the infection. The results are summarized by the author as follows:

1. The mode of reaction to infection with tubercle bacilli varies with the individual rabbit as regards the body as a whole and as regards the eyes alone.

2. In some cases there seemed to be two opposing tendencies as to the course of the disease in the body as a whole and in the eyes. In other cases the disease ran a parallel course in the body and eyes. In animals in good general condition the eyes were relatively little affected.

3. A study of the animals which were infected but not injured and of those which were injured three weeks after infection showed that the first foci appeared in the posterior part of the choroid and healed completely within from four to seven weeks.

4. Only after the posterior part of the choroid became infected did the disease spread to the anterior part. Foci were observed in the anterior part of the choroid, ciliary body and iris in all possible variations as regards order. There was complete irregularity as regards the order in which individual layers of the uveal tract were affected.

5. The manner in which the infection extended could not always be followed. In some cases it was by continuity. In others it was assumed that fresh dissemination in the eye occurred by way of the blood stream. Whether such dissemination came from a focus in the eyes or from that in another part of the body could not be determined. Cases in which there was involvement of the optic disk pointed to dis-

tribution by way of the lymphatics. In cases which revealed a layer of cells on the iris and precipitates on the posterior surface of the cornea it could be assumed that the aqueous contained organisms which were deposited on the membranes lining the anterior chamber and in this way produced the foci found. The author was unable to decide whether the bacteria found in the foci were derived from particles of tissue which had been torn off or whether they had passed directly into the anterior chamber through the walls of blood vessels.

6. In hematogenous infection with tubercle bacilli that had been grown from human blood only choroidal lesions were observed, even after a period of over six months. These lesions healed quickly, without infection of the anterior part of the uveal tract.

7. In the posterior part of the choroid no recurrence of the specific process was noted in any of the animals.

8. A fresh exogenous infection by way of the blood stream did not exercise noticeable influence on the course of the tuberculous disease in the eyes. The appearance of the tuberculous foci in the posterior part of the uvea, followed by those in the anterior part, took place in the same manner, with or without superinfection.

9. The individual forms of tissue reaction appeared, with or without superinfection.

10. The prolonged period of observation probably had more to do with the severe changes observed than had any superinfection. Whether endogenous reinfection played a rôle could not be decided.

11. The pathologic changes produced in the various parts of the eye by the experiments were extraordinarily manifold. Characteristic nodules with giant cells and necrosis were observed, as well as numerous diffuse or sharply circumscribed areas of lymphocytes and epithelioid cells in various proportions. Foci of pure noncharacteristic lymphocytes were also seen, which were interpreted to be of specific tuberculous origin. All the features of the pathologic picture often seen in spontaneous iridocyclitis in man were present, particularly infiltrations in the ciliary body, which ruptured through the pars ciliaris retinae into the vitreous. There were instances of involvement of the lymphatic spaces of the retina in the form of periphlebitis, with extension in the papilla and nerve. Deep keratitis, diffuse or circumscribed, was encountered, and also mutton fat-like precipitates on Descemet's membrane. Infiltrations in the sclera were noted.

The author invites every one who is in doubt as to the tuberculous etiology of chronic iridocyclitis in the human eye to study the lesions produced by tubercle bacilli in the eye of the rabbit. As in the human eye, the presence of tubercle bacilli in the eye of this animal soon became difficult to demonstrate.

12. The absence of typical tuberculous lesions did not mean that the eye contained no tubercle bacilli. In several cases in which histologic examination showed a nonspecific exudative inflammation, masses of tubercle bacilli were observed within the broken-down substance of the lens on staining.

13. Severe tuberculous lesions were produced in the ciliary body and iris, which did not present clinical manifestations and were recog-

nized only on histologic examination. It may be said that extensive lesions in the ciliary body may exist without symptoms, if they have not ruptured through the pars ciliaris retinae, and that lesions in the iris may remain unknown, so long as they are confined to the deep layers.

14. No conclusion could be drawn from the results of staining for tubercle bacilli as to the presence or absence of these organisms in the tissues.

15. In the phlyctenule-like formations which appeared on the eyes of animals which had been infected with organisms grown from human blood no bacilli were observed on staining. However, in a phlyctenule-like nodule on the limbus of an animal infected with a virulent organism a few tubercle bacilli were observed on staining.

16. Injury to an eye had no influence as regards the appearance of the tuberculous focus in the experimental animal.

17. Injury was responsible for the appearance of exudative iridocyclitis, which may be called posttraumatic iridocyclitis. It reached its height about fifty days after the injury.

18. The posttraumatic iridocyclitis was bound up with the tuberculous infection. When the anterior part of the uveal tract was not infected by tubercle bacilli, the site of the injury healed as smoothly as it would have done in an animal that had not been infected.

19. A latent infection in the uveal tract was found to be a contributing factor in posttraumatic iridocyclitis. This was shown by the onset of iridocyclitis when the injury had been produced only one or two days after the infection with tubercle bacilli.

20. The tuberculous process itself was, in general, not more severe in the traumatized areas of the uveal tract than elsewhere. Exceptionally, the prolapsed ciliary body was affected to a high degree.

21. The introduction of a copper splinter had no more influence on the appearance of a tuberculous process than if the injury had not been complicated by the presence of a foreign body.

22. Histologically, the copper splinter had no influence on the further development of a tuberculous process.

23. A surprising observation in the eyes with a copper splinter was the enormous increase of tubercle bacilli in the destroyed substance of the lens surrounding the splinter. At the same time, a nonspecific exudative purulent inflammation was observed histologically.

The author was stimulated to undertake these experiments by the finding of tubercle bacilli in the blood of persons with sympathetic ophthalmia (Meller). During the experiments the question repeatedly occurred whether the observations in the rabbits' eyes could be related with sympathetic ophthalmia in the human eye. Three rabbits in which the second eye became affected by iridocyclitis were studied clinically. Although the resemblance to sympathetic ophthalmia in man seemed close, the relationship was contradicted by the histologic observations. There was no doubt as to the tuberculous character of the lesions in the uninjured eyes, as caseation and tubercle bacilli were present. However, the author pointed out that these observations are not of fundamental importance, because necrosis has been seen in the human eye

in sympathetic ophthalmia, and although tubercle bacilli have not been observed as yet in histologic sections in sympathetic ophthalmia, they have been grown in culture.

In the opinion of the reviewer, these ingenious and praiseworthy experiments have been most valuable in advancing present knowledge as to what might be expected to happen if a human eye with a tuberculous lesion were subjected to injury, either surgical or accidental. The injury would have little effect on the tuberculous process, but the latter would cause posttraumatic inflammation to develop earlier and to be more severe. So far as sympathetic ophthalmia is concerned, the experiments failed to show any definite relationship between this process and tuberculosis.

BERNARD SAMUELS.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St., Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Frank H. Crawley, 5 Fitzwilliam Pl., Dublin.
Place: Bournemouth, England. Time: July 25-27, 1934.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Time: July 4-7, 1934.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Carbell, 27, Weymouth St., W. 1, London.
Place: London. Time: June 8, 1934.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Majestic Hotel, Paris. Time: May 14, 1934.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: Aug. 6-8, 1934.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. William C. Finnoff, 1612 Tremont St., Denver.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. M. Wheeler, 635 W. 165th St., New York.
President-Elect: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Sept. 9-14, 1934.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Ophthalmological Society

President: Dr. W. G. M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Lucerne, Que. Time: July 9-11, 1934.

National Society for the Prevention of Blindness

President: Mr. William Fellows Morgan.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.
Place: New York. Time: November, 1934.

SECTIONAL

New England Ophthalmological Society

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. W. Morse, 66 W. Park St., Butte, Mont.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Butte, Mont. Time: July 16-18, 1934.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Harry Shearer, 405 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. W. S. Beyer, 1101 Talcott Bldg., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. A. R. McKinney, 703 Second National Bank Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Dunbar Roy, 113 N. Pryor St., Atlanta, Ga.
Secretary: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Place: New Orleans. Time: 1934.

Southwestern Michigan Triological Society

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids.
Secretary-Treasurer: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. O. G. A. Barker, 532 Main St., Johnstown.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.
Time: Third Thursday of October and May.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 920 Republic Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Edward J. Whalen, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. W. R. Bedingfield, 753 Broad St., Augusta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.
Place: Augusta. Time: May, 1934.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Fred Overman, 611 Hume-Mansure Bldg., Indianapolis.
Place: Richmond. Time: Dec. 12, 1934.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.
Secretary-Treasurer: Dr. Lawrence A. Taylor, 118 E. Second St., Ottumwa.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Peter T. Grant, 148 Monroe Ave., Grand Rapids.
Secretary: Dr. Ralph B. Fast, 1410 Bank of Kalamazoo Bldg., Kalamazoo.
Place: Battle Creek. Time: 1934.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Gordon B. New, Mayo Clinic, Rochester.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. S. T. Hubbard, 294 State St., Hackensack.
Secretary: Dr. G. S. Laird, 125 Central Ave., Westfield.
Place: Atlantic City. Time: June, 1934.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.
Secretary: Dr. Webb W. Weeks, 20 E. 53rd St., New York.
Place: Utica. Time: May 14-16, 1934.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. O. Ruud, 318 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Valley City. Time: 1934.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Robert C. O'Neil, 105 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. C. Cody, 1304 Walker Ave., Houston.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. R. Slopanskey, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.
Secretary: Dr. Edwin W. Burton, 104 E. Market St., Charlottesville.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.
Secretary: Dr. W. F. Beckner, 1050 Fifth Ave., Huntington.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Browne Morgan, 260 Liberty St., Bloomfield.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Colin Campbell, 170 St. George St., Toronto.
Secretary: Dr. H. M. Macrae, 170 St. George St., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. L. McDougall, 50 Whitehall St., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Benjamin Rones, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. P. Chamberlain, 7405 Detroit Ave., Cleveland.
Secretary: Dr. A. D. Ruedemann, Cleveland Clinic, Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. M. Goldberg, 328 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. T. S. Love, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Daniel Brannin, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. C. Walker, 410 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. Edward B. O'Keefe, 901 State St., Schenectady.
Secretary-Treasurer: Dr. W. C. Mott, 118 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. E. L. Howard, 600 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. E. L. Goar, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. J. C. Dickson, 622 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. D. A. Bartley, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. M. B. Simpson, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Clifford S. Losey, 219 E. 10th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. H. Brandt, 1136 W. Sixth St., Los Angeles.
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Charles Dwight Townes, 332 W. Broadway, Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, 1103 Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. George B. Trible, 1801 I St., Washington.
Secretary: Dr. Harry F. Davies, 1835 I St., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
President: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Frederick E. Hasty, 119, 7th Ave. N., Nashville, Tenn.
Secretary-Treasurer: Dr. Kate Savage Zerfoss, 803 Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Webb William Weeks, 20 E. 53rd St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Abbott M. Dean, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: Second Tuesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Lee, 501 E. Franklin St., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. W. D. Edwards, 389 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. E. C. Spitze, 234 Collinsville Ave., East St. Louis, Ill.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. King Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert S. Irvine, 490 Post St., San Francisco.
Secretary: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

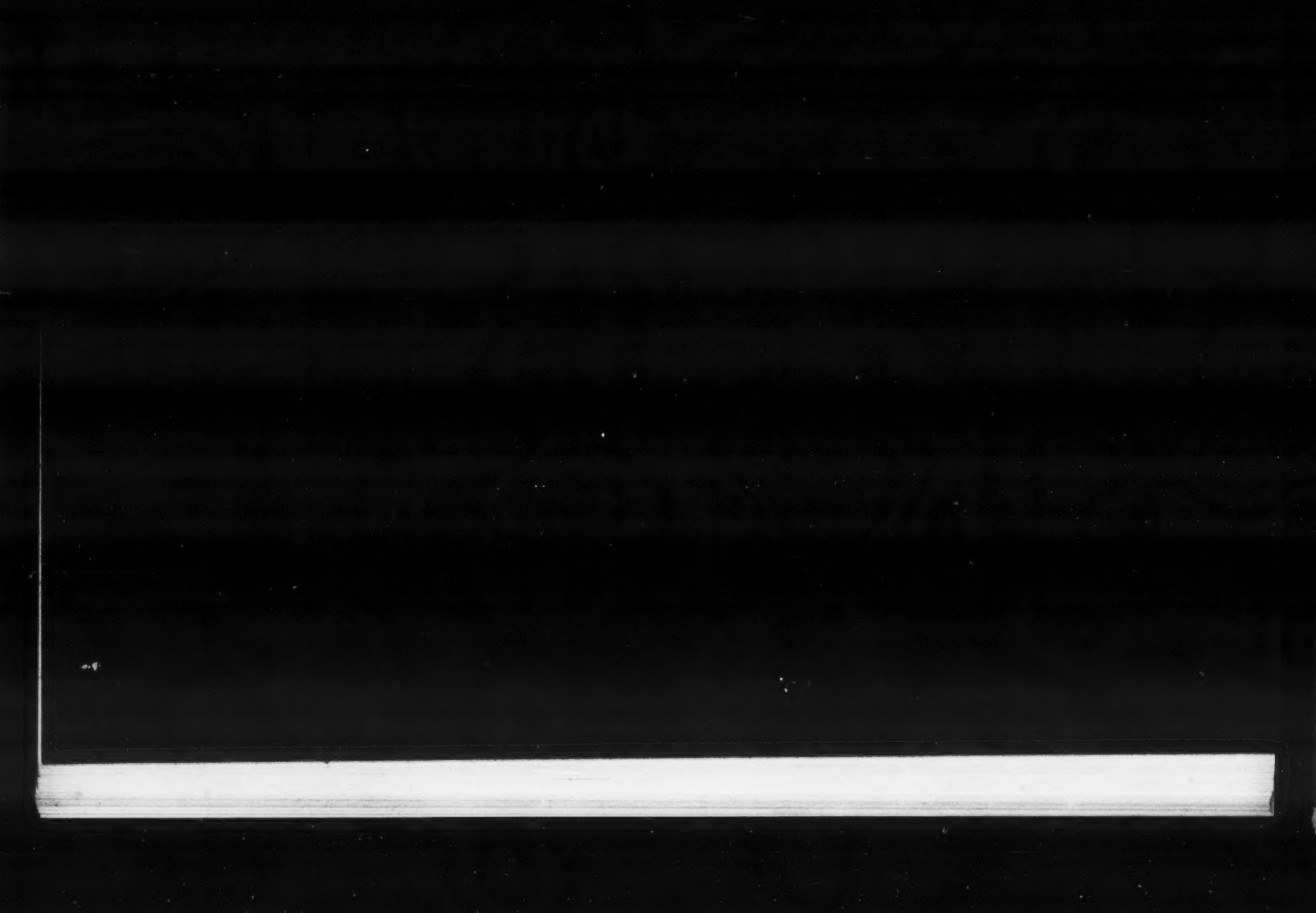
President: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Secretary-Treasurer: Dr. Leon F. Gray, 1214 Slattery Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Marc Anthony, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. Raymond A. Lower, 1455 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. H. O. Brust, 820 S. Crouse Ave., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelmann, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, Medical Arts Bldg., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.